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Asthma rates clustered in families, were higher in male children and female parents, and were comparable to other studies. Significant increases of lower respiratory disease were also found among asthmatic children in the high exposure community. Difference in parental recall, family size, or parental cigarette smoking were not likely explanations for the excess morbidity in the high exposure community. Therefore, these results associate excess acute lower respiratory disease in children with exposure to elevated particulate levels and low sulfur dioxide concentrations.

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FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the public health and welfare and the productive capacity of our Nation's population.

The Health Effects Research Laboratory, Research Triangle Park, conducts a coordinated environmental health research program in toxicology, epidemiology, and clinical studies using human volunteer subjects. These studies address problems in air pollution, non-ionizing radiation, environmental carcinogenesis and the toxicology of pesticides as well as other chemical pollutants. The Laboratory develops and revises air quality criteria documents on pollutants for which national ambient air quality standards exist or are proposed, provides the data for registration of new pesticides or proposed suspension of those already in use, conducts research on hazardous and toxic materials, and is preparing the health basis for non-ionizing radiation standards. Direct support to the regulatory function of the Agency is provided in the form of expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of health care and surveillance of persons having suffered imminent and substantial endangerment of their health.

Pollution of the ambient air is complex. Many substances are emitted into the air from a variety of sources. They differ physically, being gases, vapors, droplets and particles of many sizes and shapes. They also differ chemically, some being very irritating, some odorous, some neutral. They may change considerably after they are emitted into the air, sometimes forming other compounds. In order to understand the relative importance of each kind of pollutant in producing effects on health it is necessary to perform studies where that pollutant is dominant in relation to others with which it is commonly associated. This study attempts to examine the effects of high particulate pollution where sulfur oxides pollution is at a low level.

John H. Knelson, M.D.
Director,
Health Effects Research Laboratory

ABSTRACT

Acute lower respiratory disease was surveyed by questionnaire among parents of 10,000 children aged 1 to 12 years in two Southeastern communities representing intermediate and high exposures to particulates and low sulfur dioxide levels. Morbidity reporting patterns with respect to age, parental education, and history of asthma were similar for blacks and whites, but the frequency of pneumonia was significantly lower, and the frequencies of croup, bronchitis, and "any lower respiratory disease" were significantly higher among whites in both communities. Significant increases of any lower respiratory diseases and hospitalization were found among children in the high exposure community.

Asthma rates clustered in families, were higher in male children and female parents, and were comparable to other studies. Significant increases of lower respiratory disease were also found among asthmatic children in the high exposure community.

Differences in parental recall, family size, or parental cigarette smoking were not likely explanations for the excess morbidity in the high exposure community. Therefore, these results associate excess acute lower respiratory disease in children with exposure to elevated particulate levels and low sulfur dioxide concentrations.

This report covers a period from 1960 to 1971 and work was completed April 1976.

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When I first became interested in research and in epidemiology, I dreamt of discovering things never before known. Now, years later, I still dream of discovery, but the times I have been fortunate enough to scale a peak and glimpse some unknown, I have realized that others before me oft had looked out from the same place. It doesn't impair the view; it just keeps you honest. Doctors and scientists and other grownups of that ilk spend too little of their time thanking others who helped them learn and grow. Too bad, because we might occasionally remember that the world was running before we got here. It is never possible to mention everyone -- but thanks to the faculty of the Harvard School of Public Health and particularly to the members of my thesis committee, Drs. Jacob F. Feldman, Benjamin G. Ferris, Jr., and George B. Hutchinson; to Jim Stebbings, Fred Miller, Andy Stead, Dennis House, Kathryn McClain, and Carol Riggs of the United States Environmental Protection Agency, and lastly to my parents, who always helped me learn and grow.

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SECTION 1

INTRODUCTION

Acute respiratory diseases are the most common illnesses in children, and those of the lower respiratory tract may well portend chronic respiratory disease in later life.[1-3] The lower respiratory tract is commonly defined as the portion of the respiratory tract beginning at the larynx and extending out to terminal alveoli therefore including the trachea, bronchi, bronchioles and lung parenchyma, and stroma. In the Harvard longitudinal studies of child health and development, 83% of all illnesses experienced from birth to 18 years of age were respiratory tract infections.[4] In the survey of "One Thousand Families in Newcastle-upon-Tyne," 53% of all illnesses in the first five years involved the respiratory tract.[5] Unlike adults, elementary school children are generally not exposed to occupational pollution or self-pollution by cigarette smoking. Children are also less likely to have experienced a variety of complicated long term ambient air pollution exposures related to residential mobility. Hence, they are an excellent group in which to study adverse health effects associated with community air pollution.

Studies in England, Japan, and Russia have implicated sulfur dioxide and particulates in the ambient air as a cause of increased respiratory morbidity in children.[6-10] Studies in New York and Chicago found increased acute respiratory morbidity associated with elevated exposure to combined sulfur oxide and particulate air pollution among parents and their children.[11] Recent retrospective surveys of acute lower respiratory disease in children living in smelter communities in Utah and the Rocky Mountains found excess bronchitis and croup, but not pneumonia or hospitalization, associated with elevated sulfur oxide exposures lasting three or more years.[12,13] None of these studies was able to distinguish the effects of individual air pollutants such as sulfur dioxide or particulate matter from the more complex urban mixtures. A more detailed review of the relevant literature is appended (Appendix A).

Since air pollution control technology is often directed towards single pollutants, it is especially important to disentangle the effects of exposure to multiple pollutants. For example, none of the above studies could assess the health effects of exposure to total suspended particulates in the presence of relatively low levels of sulfur dioxide. Yet such an ambient pollutant pattern is typical of industrial Birmingham, Alabama, where low sulfur coal has been used for many years. This report describes a retrospective survey of frequency of acute lower respiratory illnesses among elementary school children in two Southeastern U.S. communities. The primary study hypothesis was that reported respiratory morbidity rates would be higher in Birmingham, the high pollution exposure community, than in Charlotte, the intermediate exposure community.

Children with asthma are known to have a high frequency of lower respiratory tract infections.[14] In two community morbidity surveys in the western United States,[12,13] children with a history of asthma reported more than twice as much bronchitis, croup, pneumonia, and hospitalization for any of these diseases when compared to nonasthmatic children. Furthermore, croup, bronchitis, and pneumonia rates were significantly increased among asthmatic children residing in the communities with higher exposures to sulfur oxides, suspended sulfates, and particulate matter.

Bronchial asthma is characterized by periodic attacks of obstructive expiratory dyspnea of variable severity, duration and frequency.[15] Pathophysiologically, recent work suggests obstruction of large as well as small airways and that the disease is not solely immunologic in nature.[16] Persons with asthma were affected much more frequently than nonasthmatics during the acute smog episodes of Donora and London.[17,18] Much evidence since then has associated increased attacks of asthma with exposure to ambient air pollution.[19,20] Hospitalization for asthma among children under 15 years of age was found to be related to particulate air pollution exposure in Erie County, New York.[21] A more detailed review of the relevant literature is cited in Appendix A.

Excessive acute lower respiratory disease in nonasthmatic children has been shown to be associated with exposure to total suspended particulate matter in the presence of low sulfur dioxide levels in a recent study in Birmingham, Alabama, and Charlotte, North Carolina.[22] This report presents a detailed account of the prevalence of asthma in families and the frequency of acute lower respiratory disease in children with a history of asthma. The primary hypotheses were (1) reported morbidity rates would be positively related to a history of asthma in children, and (2) reported morbidity rates among asthmatic children would be highest in Birmingham, the higher pollution exposure community.

SECTION 2

CONCLUSIONS

In this study, reported acute lower respiratory disease in children was found to be related to elevated total suspended particulate exposure, to a history of asthma, to the education of the head of the household, and to race. This study has suggested that exposure to elevated concentrations of total suspended particulate matter and suspended sulfates in the presence of extremely low sulfur dioxide concentrations does indeed increase the risk of acute lower respiratory disease in children. Morbidity excesses in Birmingham were found among both black and white children without a history of asthma. Among black children, significant increases in Birmingham were found for one or more episodes of "any lower respiratory disease", croup, pneumonia, and hospitalization, but not bronchitis (Table 1). In general, two or more episodes of all morbidity conditions did not differ significantly by community among black children. Among white children, statistically significantly increases in Birmingham were found for one or more episodes of all reported morbidity conditions (Table 1). Similar results were found for two or more episodes of all morbidity conditions. No statistically significant differences were found in which morbidity rates in Charlotte exceeded those in Birmingham among either black or white children without a history of asthma. These results appear strengthened by the finding of decreased pulmonary function among black and white school children in Birmingham compared to those in Charlotte.[23]

Although one would certainly expect asthmatic children to be at least as sensitive to air pollution as nonasthmatic children, the findings with regard to air pollution were less clear for asthmatic children. Among children with a history of asthma, morbidity rates were higher in Birmingham in half of the comparisons studied. Croup rates were significantly increased in Birmingham among black and white children although those for bronchitis ($0.10>p>0.05$) were higher in Charlotte among black children.

Several factors may have been related to the weaker intracommunity differences found among asthmatic children. First, other environmental exposure factors than air pollution, such as temperature, dusts, pollens, and other allergens were not measured in this study. Although these factors have been associated with increased acute asthmatic attacks, and not increased acute lower respiratory morbidity, they may also increase the risk of lower respiratory disease in asthmatic children. If the latter is true, the children's exposure to these factors was not estimated and may have been quite different from their estimated exposure to ambient air pollution. A second and more conjectural reason is related to the relatively high frequency of respiratory morbidity among all asthmatic children regardless of community or race, viz., the risk associated with a history of asthma may be a much stronger determinant for lower respiratory disease than exposure to community air pollution. If this were true, the effects of air pollution upon lower respiratory disease would be relatively less, and more difficult to determine statistically. There is some evidence for this hypothesis in that the relative black/white differences generally were least among asthmatic children (Table 2). A third factor is the considerably reduced statistical power to determine any true intracommunity differences which is due to the small sample size of the children with a history of asthma.

Respiratory morbidity risk was found to be related to a child's history of asthma, as expected. In general, all respiratory morbidity was lowest in children without a history of asthma, intermediate in children with asthma diagnosed by a doctor, but presently inactive, and highest in children with a history of presently active asthma for both blacks and whites (Table 2). When compared to nonasthmatic children, those with active asthma reported from 2.5 to almost 4 times as much "any lower respiratory disease." Furthermore, a much higher proportion of asthmatic children had repeated episodes of respiratory moribidty. These results provide quantitative estimates for common clinical experience. Indeed, they underscore the recognized need for vigorous and vigilant medical care for asthmatic children.

Education of the head of the household and sex, in addition to age and air pollution exposure, were found to be determinants of morbidity reporting. "Any lower respiratory disease" and bronchitis were reported more frequently among white males, but no statistically significant differences with regard to sex were found among black children. "Any lower respiratory disease" and bronchitis were found to be increased in both black and white children from households with a high school or greater education. Two or more episodes of croup did not vary significantly by education of the head of the household. It was not possible to properly interpret the statistical relationships of one or more episodes of croup, pneumonia, and hospitalization to education of the head of the household, per se, for blacks and whites. This was because either a significant "city x SES" (SES, socioeconomic status) or a "city x age x SES" interaction was found for all three of these conditions in the saturated linear model for categorical data, and the reduced models were directed towards examining the "city" effect rather than the "SES" effect (cf. Tables 3 and 4).

In two other published studies which used this questionnaire, similar results were reported, viz., statistically significant more frequent "any lower respiratory disease," and bronchitis (and croup) in children from households with a high school or greater education and the converse for pneumonia and hospitalization.[12,13] However, the specific form of the linear model for categorical data used for statistcal analyses in both of these studies was not discussed. Statistical summaries in both papers imply the following model: city/pollution, age, sex, and SES. However, there is some question of the validity of the previous findings regarding the relationship of croup, pneumonia, and hospitalization to socioeconomic status, since this study found several significant interactions involving city/pollution and SES when a saturated model was used (cf. Appendix B). Nevertheless, we should learn more about reported lower respiratory disease morbidity in relation to medical care availability and medical care utilization. The

British-United States differences with respect to childhood lower respiratory disease and social class suggest that differences in the availability of medical care may be involved.

Although both sex and education of the head of the household were found to be determinants of morbidity reporting, the effect of sex was found only for "any lower respiratory disease" and bronchitis among white children. However, reporting of "any lower respiratory disease" and bronchitis was related to education of the head of the household among children of both races. For any given morbidity condition in which they were both statistically significant, the relative increase for education of the head of the household was about the same as the relative increase for sex as evidenced by the example in Table 5. Age and sex distributions would be most likely to be comparable between communities when sampling through the elementary schools. However, failure to ascertain education of the head of the household (or some other good index of socioeconomic status) could easily lead to confounding intracommunity differences due to air pollution with those due to socioeconomic status in a study of this type.

When compared to white children, black children reported more pneumonia, but less of all four other morbidity conditions (Table 6). This was true for both communities as well as for all categories of asthma history. A study in New York City using a similar questionnaire has confirmed these findings.[24] Yet in both this and the New York study, excess respiratory morbidity in black or white children was associated with exposure to sulfur oxide and particulate matter despite intracommunity black/white morbidity differences. In addition to the effects of air pollution, respiratory morbidity in children of both races showed similar patterns with regard to age, sex, education of the head of the household, and a history of asthma.

Differences in medical care utilization could explain, in part, the observed differences in morbidity reporting by race. One may assume that sicker children are brought to physicians more frequently by their parents and this would be largely independent of other determinants such as race. In this study, the facts that black/white morbidity differences were least among children with active asthma, and that pneumonia (a serious illness and often a consequence of prior, unattended, milder upper or lower respiratory disease) was reported most frequently among blacks support this assumption. Further, if the black/white morbidity differences are greatest among less seriously ill children, this suggests that the observed racial differences would be most easily explained by differences in cultural and socioeconomic factors, rather than genetic factors, per se. At any rate, the reasons for the observed black/white morbidity differences appears to be a fertile area of research from both the scientific and public health viewpoint.

The questionnaire used in this study has been used twice previously in reported studies.[12,13] This study has shown that the increased risk of acute lower respiratory disease in children is related to the history of asthma activity as well as a history of asthma per se. Children with a history of asthma should be considered separately when estimating the true risk of acute lower respiratory disease in children although their inclusion would confound intercommunity comparisons only if asthma prevalence was considerably higher than usually observed, and in addition, higher in one community than the other. Although several findings require further study, the agreement of asthma prevalence in families with findings in several other reports, the increased risk of morbidity among asthmatic children, and the consistent relationships of lower respiratory disease morbidity in nonasthmatic children with age, sex, and parental education found in blacks and whites testify to the utility and reliability of this questionnaire for community surveys. Further discussion on the validity of the questionnaire is appended (Appendix C).

It appears quite likely that elevated exposure to suspended particulate matter (total and sulfate fraction) without concomitant sulfur dioxide exposure is sufficient to increase childhood respiratory morbidity. All children were affected by exposure to air pollution although the effect of this exposure varied with race, sex, and socioeconomic status. Much of the suspended sulfate fraction may represent sulfur dioxide which has sorbed onto particles. In fact, it would be desirable to further characterize the physical and chemical aspects of ambient suspended particulate matter to gain a better understanding of the toxicology of these substances. Obviously, the use of individual pollutant concentrations, as opposed to some product or combination thereof, is a relatively simple way to estimate human exposure to air pollutants. Nevertheless, one cannot deny that these simple indices have been quite useful epidemiologically in many studies all over the world. Epidemiologic associations with individual pollutant indices are much to be preferred for regulatory purposes as most control strategies are directed toward individual pollutants.

Epidemiologic studies relating health effects to long term exposure to ambient air pollutant concentrations must make a concerted effort to estimate past exposures in addition to a current monitoring. If ambient pollutant levels have remained constant in an area, no error is incurred. If pollutant concentrations have been decreasing with time, attributing the health effect to current levels imposes an unnecessary and possibly severe economic penalty. Conversely, when pollutant concentrations have been increasing with time, attributing the health effect to current levels fails to fully protect the public health. Two important questions are of related interest: First, to what extent is exposure to intermittent peak levels, as opposed to less variable lower level exposures, the cause long term health affects? Studies in smelter communities, may provide some answers since they are exposed to frequent short term fumigations and yet they often have annual pollutant averages near Federal standards. Second, how long does it take for excess morbidity to decrease after pollution is controlled in a community? It will require some time to obtain answers to both of these questions.

Respiratory diseases were a common cause of morbidity and mortality in Colonial America and are still the most common childhood illnesses in the United States today.[25] Children experience about five or six acute respiratory illnesses yearly and many of these will involve the lower respiratory tract.[1,2,26,27,28] Mortality due to acute lower respiratory disease is a serious problem in children under five years of age.[14,29,30,31] Recent evidence has strengthened the hypothesis that frequent episodes of lower respiratory disease in childhood are associated with the development of chronic respiratory disease in later life.[3,14,32] Although the relative risk of childhood acute lower respiratory disease from exposure to sulfur oxides and particulate air pollutions ranges from about 1.2 to 2.0, the attributable risk becomes enormous when one considers the acute misery, the interference with school (and parental work) activities, the family medical costs, the increased burdens on the medical care system, and the real possibility of an increased risk of chronic respiratory disease in later life for children so exposed.

TABLE 1. FOUR YEAR FREQUENCY OF ONE OR MORE EPISODES OF EACH MORBIDITY CONDITION: MODEL ADJUSTED RATES* FOR CHILDREN AGED 1 TO 12 YEARS

Race	Community	Any LRD		Croup		Bronchitis		Pneumonia		Hospitalization	
		Female	Male	<HS	≥HS			<HS	≥HS		
Black Children	Charlotte	16.6%	18.4%	7.4%	5.7%	9.2%		9.5%	7.8%	Second	
	Birmingham	21.0%	19.2%	6.5%	8.2%	8.4%		11.8	13.4%	Order	
	Birmingham/Charlotte	1.27	1.04	0.88	1.44	0.91		1.24	1.72	Interaction	
						Female	Male			<HS	≥HS
White Children	Charlotte	28.7%		Second		20.3%	18.2%	Second		3.1%	2.5%
	Birmingham	33.6%		Order		22.9%	25.0%	Order		4.1%	4.8%
	Birmingham/Charlotte	1.17		Interaction		1.13	1.37	Interaction		1.32	1.92

*Rates for nonasthmatic children with three or more years residence duration from saturated linear model for categorical data and adjusted for variable(s) not displayed (age, sex, or education of head of household).

TABLE 2. CRUDE RATES OF ONE OR MORE EPISODES OF "ANY LOWER RESPIRATORY DISEASE" IN RELATION TO A HISTORY OF ASTHMA DIAGNOSED BY A DOCTOR

City	History of Asthma	"Any Lower Respiratory Disease", Crude Rate*		Ratio Black/White
		Black	White	
Charlotte	Never Diagnosed	16.2%	27.1%	1.67
	Diagnosed, Inactive	42.4%	55.6%	1.31
	Diagnosed, Active	62.8%	83.5%	1.33
Birmingham	Never Diagnosed	18.8%	31.8%	1.69
	Diagnosed, Inactive	48.6%	47.7%	0.98
	Diagnosed, Active	60.0%	80.0%	1.35%

*Crude rate of children aged 1 to 12 years with three or more years residence duration.

TABLE 3. CHI-SQUARE AND SIGNIFICANCE LEVELS FOR FACTORS AFFECTING LOWER RESPIRATORY DISEASE AS DETERMINED BY A SATURATED LINEAR MODEL FOR CATEGORICAL DATA: BLACK CHILDREN WITH THREE OR MORE YEARS OF COMMUNITY RESIDENCE

Effect	Degrees of Freedom	Any LRD		Croup		Bronchitis		Pneumonia		Hospitalization	
		≥1	≥2	≥1	≥2	≥1	≥2	≥1	≥2	≥1	≥2
City/Pollution (P)	1	0.79	0.09	0.02	0.01	0.60	0.71	8.12^{b}	0.99	2.50	Rate too low to fit model
Age (A)	2	35.14^{a}	16.34^{a}	13.47^{b}	3.39	13.81^{a}	8.09^{c}	6.92^{a}	1.58	12.00^{b}	
Sex (S)	1	<0.01	0.49	0.12	0.34	0.96	0.06	1.09	0.01	0.53	
SES (E)	1	5.53^{c}	8.55^{b}	2.88^{d}	1.08	8.76^{b}	3.57^{d}	0.56	1.89	0.26	
City x Age	2	3.33	7.30^{c}	1.11	7.49^{c}	1.72	2.30	1.76	3.67	0.15	
City x Sex	1	3.56^{d}	5.79^{c}	2.23	1.43	1.63	0.73	0.96	0.73	0.48	
City x SES	1	0.05	4.94^{c}	2.72^{d}	0.14	0.96	0.04	3.20^{d}	1.07	0.41	
Age x Sex	2	0.91	1.46	0.45	1.08	1.32	5.48^{d}	1.56	0.32	6.96^{c}	
Age x SES	2	3.49	5.95^{c}	1.00	2.21	3.71	3.02	4.07	0.77	0.05	
SES x Sex	1	0.29	0.02	0.31	0.16	1.81	1.90	0.46	0.46	0.64	
P x S x A	2	4.22	2.72	4.27	0.57	1.71	1.96	0.85	0.79	0.69	
P x E x A	2	0.04	1.17	0.28	2.12	2.12	1.31	1.35	0.18	6.36^{c}	
P x E x S	1	<0.01	0.03	0.02	1.53	0.37	0.02	0.01	0.08	1.95	
A x S x E	2	2.63	0.46	0.75	0.76	0.63	2.96	2.48	0.11	4.87^{d}	
P x A x S x E	2	2.20	0.50	1.71	1.05	0.16	2.33	0.97	0.81	1.56	

a - $p \leq 0.001$
b - $p \leq 0.01$
c - $p \leq 0.05$
d - $0.10 > p > 0.05$

For each term in the model, the probability for a two-tailed test of statistical significance.

TABLE 4. CHI-SQUARE AND SIGNIFICANCE LEVELS FOR FACTORS AFFECTING LOWER RESPIRATORY DISEASE AS DETERMINED BY A SATURATED LINEAR MODEL FOR CATEGORICAL DATA: WHITE CHILDREN WITH THREE OR MORE YEARS OF COMMUNITY RESIDENCE

Effect	Degrees of Freedom	Any LRD		Croup		Bronchitis		Pneumonia		Hospitalization	
		≥1	≥2	≥1	≥2	≥1	≥2	≥1	≥2	≥1	≥2
City/Pollution (P)	1	9.91^{b}	10.99^{a}	0.74	4.59^{c}	11.99^{a}	24.18^{a}	3.43^{d}		5.53^{c}	
Age (A)	2	56.52^{a}	31.59^{a}	22.83^{a}	7.08^{c}	39.69^{a}	18.10^{a}	10.95^{b}		36.61^{a}	
Sex (S)	1	4.40^{c}	4.83^{c}	0.65	0.09	6.24^{b}	8.40^{b}	0.01		0.77	
SES (E)	1	8.43^{b}	4.44^{c}	3.57^{d}	0.71	12.54^{a}	7.22^{b}	0.97	Rate	8.15^{b}	Rate
City x Age	2	0.61	0.06	2.96	4.09	0.71	1.37	0.32	too	3.48	too
City x Sex	1	1.21	1.51	0.32	0.43	3.21^{d}	2.04	0.91	low	0.71	low
City x SES	1	<0.01	0.01	0.14	0.09	2.11	0.03	0.09	to	3.88^{c}	to
Age x Sex	2	4.18	2.41	4.55	4.40	1.90	3.03	1.74	fit	1.22	fit
Age x SES	2	2.32	0.43	4.24	2.18	0.81	0.47	1.24	model	3.44	model
SES x Sex	1	0.27	1.77	2.23	0.21	0.04	0.35	2.12		0.07	
P x S x A	2	0.72	0.53	0.89	1.03	0.47	2.33	4.48		2.54	
P x E x A	2	2.08	1.56	7.78^{c}	1.96	4.16	0.76	8.37^{c}		2.59	
P x E x S	1	0.24	0.75	0.24	2.12	1.11	0.58	1.25		0.49	
A x S x E	2	0.71	4.28	2.13	1.80	1.29	3.51	3.07		0.39	
P x A x S x E	2	4.74^{d}	3.56	5.40^{d}	5.70^{d}	2.60	2.32	2.41		1.59	

a - $p<0.001$
b - $p<0.01$
c - $p<0.05$
d - $0.10>p>0.05$

For each term in the model, the probability for a two-tailed test of statistical significance.

TABLE 5. FOUR YEAR FREQUENCY OF "ANY LOWER RESPIRATORY DISEASE" AND BRONCHITIS IN WHITE CHILDREN BY SEX AND EDUCATION OF THE HEAD OF THE HOUSEHOLD

Morbidity Determinant		Model Adjusted Rates*	
		≥2 "Any LRD"	≥2 Bronchitis
Sex	Female	17.3%	9.9%
	Male	19.1%	12.1%
	Male / Female	1.10	1.22
Education, Head of the Household	<HS	16.8%	9.2%
	>HS	19.6%	12.7%
	>HS / <HS	1.17	1.38

*Age-city-education head of household (for sex) or age-city-sex adjusted rates for nonasthmatic children with three or more years residence duration, from saturated linear model for categorical data (Table 4).

TABLE 6. CRUDE RATES FOR ONE OR MORE EPISODES OF ALL MORBIDITY CONDITIONS, BY CITY AND RACE*

Morbidity Condition	City	Crude Rates		Ratio White/Black
		Black	White	
"Any lower respiratory disease"	Charlotte	16.2%	27.1%	1.67
	Birmingham	18.8%	31.8%	1.69%
Croup	Charlotte	6.5%	12.4%	1.91
	Birmingham	6.9%	14.4%	2.09
Bronchitis	Charlotte	7.9%	18.4%	2.33
	Birmingham	7.8%	23.5%	3.01
Pneumonia	Charlotte	8.4%	6.3%	0.75
	Birmingham	12.2%	7.7%	0.63
Hospitalization	Charlotte	1.9%	2.3%	1.21
	Birmingham	3.2%	4.5%	1.41

*Restricted to children without a history of asthma and with three or more years of community residence.

SECTION 3

RECOMMENDATIONS

Very little research has been done which makes fine distinctions of the effects of single pollutants on the lower respiratory tract. This study was to gather data on lower respiratory infection in children exposed to suspended particulates in the presence of low levels of sulfur oxides.

The reasons for increased morbidity rates for whites, except for pneumonia (as shown in Table 6) should receive further study to determine valid explanations. Also, the chemical, physical and toxicological properties of the ambient suspended particulate matter should receive further study.

SECTION 4

MATERIALS AND METHODS

Community Selection

Two communities in the southeastern United States were selected on the basis of past air quality data and historical information regarding pollutant emissions. The communities were ranked intermediate or high on the basis of the estimated exposure to total suspended particulates during the period covered by the study and for the prior decade. The exposure rankings were relative for the two cities and not quantitatively related to the current U.S. National primary standard for particulate matter. Charlotte, the intermediate exposure community with a population of over 240,000, is a growing commercial and light industrial center in the Piedmont region of North Carolina. Located in Mecklenberg County, its major industries include chemicals, textiles, fabricated metals, machinery, wood, foundries, cement, and asphalt. When compared to Birmingham, Alabama, Charlotte had both lower emissions of particulates and less frequent temperature inversions. Birmingham, Alabama, the high exposure community, with a population of over 300,000, is one of the nation's leading industrial centers. It is, perhaps, best known for its steel production and manufacturing. However, it also produces fabricated metals, transportation equipment, machinery, stone, clay, glass, and wood products. Industrial plants, located throughout the city, are surrounded by commercial and residential areas.

Assessing Air Pollution Exposure

At the time of this study, air monitoring stations were located within each community within 1 1/2 to 2 miles of the study population. All stations had their air inlet six feet above the ground except for one which was 16 feet above the ground. At each station, 24-hour integrated samples of sulfur dioxide (modified West-Gaeke method), total suspended particulates (high-volume samplers), suspended sulfates, and suspended nitrates were monitored on a daily basis. Dustfall was determined from monthly samples. Estimates of past exposure were derived from previous aerometric data and emissions data. A full description of the location of monitoring stations and the methodology for current data collection and estimation of past exposures has been presented elsewhere (available from EPA upon request).[33] Section 5 provides further detail regarding air pollution exposure.

Collection of Health and Demographic Data

In November 1971, elementary school children (grades 1 to 6) in selected study communities in each city were asked to take explanatory letters and questionnaires home to their parents. Socioeconomically similar school districts in each city were selected by consultation with the Superintendent of Schools and school principals. Children in eleven schools in Birmingham and twelve schools in Charlotte were surveyed. The frequency of acute lower respiratory disease was ascertained by means of a School and Family Health Questionnaire, which was completed by the mother or female guardian in each family when possible and returned to the school (Appendix E). Mothers were asked to answer the questions about lower respiratory disease for all children twelve years of age or younger who lived in the household.

The questionnaire inquired about the frequency of treatment by a physician for pneumonia, croup, or bronchitis (including bronchiolitis or deep chest infections other than pneumonia or croup) during the period beginning in September 1967 and continuing through the time of questionnaire completion. Hence, all morbidity rates presented encompass a time period of just over four years. Other information ascertained included hospitalizations for lower respiratory illnesses, name of children's current physician, history of asthma diagnosed by a doctor, length of residence in the community, number of living quarter changes, education of head of household as an index of socioeconomic status, parents' smoking statuses, race, family census, number of rooms per household and presence and type of air conditioning per household. In Birmingham, respondents with telephones were called if any information was missing from their questionnaire. In Charlotte, respondents with telephones were *not* called if the following information was missing: education of head of household, race, number of living quarter changes, number of rooms per household, presence and type of air conditioning, history of asthma diagnosed by a doctor and age of mother or female guardian; they *were* called if any of the other information was missing.

Data Analysis and Hypothesis Testing

As planned in the first part of this protocol, children with a history of asthma, and all children with less than three years residence in the community, were excluded from the analysis. Children with asthma are known to have a higher risk of respiratory illnesses than nonasthmatic children. Asthma history was determined by "yes" or "no" answers to the following two questions: (1) "Has this person ever had asthma diagnosed by a doctor?" and (2) "Has this asthma been active in the past two years?" As a matter of scientific interest, these differences were studied and the results are shown separately.[22,34,35] Children with less than three years residence were excluded for several reasons. They had been exposed to the community air for only a short time and their previous residences and pollutant exposures were not known. Moreover, illness itself has been associated with migration, and recently migrated families would be less likely to have established patterns of medical care within a community, possibly obscuring the effect of pollution on reported doctor-diagnosed illnesses within a community. (For blacks, morbidity rates were generally higher among recent migrants; for whites morbidity rates were generally higher among residentially stable children. This study was not designed to explain these differences.) Children under one year of age were excluded because of an error in coding instructions which made children under one year indistinguishable from children with missing information on age.

Actual or direct-adjusted rates are presented for descriptive purposes, for comparison to other published data and for comparison to the model-adjusted rates derived from the linear categorical model described below. Descriptive morbidity rates (as in Tables 7-10) were direct-adjusted according to standard methods, the reference populations being all nonasthmatic children with three or more years residence.[36] City questionnaire return rates in Table 2 were tested by standard contingency table techniques.[37]

The primary test hypothesis was that the frequency of reported physician treatment for "any lower respiratory disease," croup, bronchitis, pneumonia and hospitalization, appropriately adjusted, would correspond to the gradient in pollutant exposures, viz. that respiratory morbidity and related hospitalization rates would be higher in Birmingham than in Charlotte. The combined disease category, "any lower respiratory disease," was constructed to include a child if he had either pneumonia, croup, or bronchitis or any other deep chest infection. Hence, the category "any lower respiratory disease (any LRD)" is an index of overall frequency of acute lower respiratory disease in the community without regard to specific diagnostic categories. For each of the five reported conditions, two sets of analyses were done. In the first, the dependent variable was the percent of children reporting one or more episodes of each morbidity condition. In the second, the dependent variable was the percent of children reporting two or more episodes of each condition.

For nonasthmatic children, each specific hypothesis was tested statistically in a general linear model for categorical data.[38] An alpha probability of $p<0.05$ was chosen as statistically "significant." All statistical tests were "two-tailed" tests, i.e., for any given term in the model, such as "city/pollution," the resulting chi-square and associated probability were for excesses in either city as large or larger than those observed, given that there is no difference. The general linear model for categorical data technique utilizes weighted regression on categorical data and allows estimation of each individual factor adjusted for all other factors in the model.

For asthmatic children, each specific hypothesis was tested by standard contingency table techniques.[37,39] An alpha probability of $p < 0.05$ was chosen as statistically "significant." All statistical tests were "two-tailed" tests, i.e. the resulting chi-square and associated probability were for excesses in either city as large or larger than those observed, given that there is no difference. Morbidity rates in children with a history of asthma active within the past two years ("active") were generally higher than those with a history of asthma, but not active within the past two years ("inactive"). Therefore rates for each group were compared separately between cities, e.g. morbidity rate for inactive asthmatic children in Charlotte to the comparable rate for inactive asthmatic children in Birmingham. Because of the considerably smaller sample sizes of both categories of asthmatic children it was not possible to adjust their morbidity rates for age, sex, and education of the head of the household (SES) while testing for "city" differences. Hence the crude rates of each morbidity condition among children aged 1-12 years were used for statistical testing. This was not unreasonable since active and inactive children were comparable with regard to age, sex, and SES distributions. Likewise, the crude morbidity rates of nonasthmatics were used for descriptive purposes unless otherwise noted.

Morbidity conditions were analyzed in a saturated analysis of variance (ANOVA) form of the linear model, namely, four main effects: city/pollution (P), age (A), sex (S), education of head of household (E), and all possible interactions (six first-order, four second-order, and one third-order). For each morbidity condition analyzed in the model, *age* (1 to 4, 5 to 8, 9 to 12), *sex* (F-female; M-male), and *education of the head of the household* (<HS - less than high school, ≤ HS - high school or more) as an index of socioeconomic status (SES), were considered as intervening variables, and *city/pollution* (C-Charlotte, B-Birmingham) as the independent variable of primary interest. Two or more episodes of hospitalization among both races and two or more episodes of pneumonia among whites were not tested statistically because their rates were too low to obtain reliable estimates from the model. Therefore, they were analyzed descriptively and discussed in context with the results of the statistical tests. Statistically significant interaction terms involving "city/pollution" were explored further to account for them. ANOVA models to account for statistically significant "city/pollution" interaction terms are presented and discussed in the statistical appendix (Appendix B). Observed initial reported rates for each morbidity condition and number of episodes among nonasthmatic black and white children with three or more years residence duration by city, age, sex, and SES are submitted in Appendix D.

TABLE 7. FOUR YEAR REPORTED RATES OF ONE OR MORE EPISODES OF EACH MORBIDITY CONDITION AMONG BLACK CHILDREN, BY COMMUNITY

		Direct Adjusted* Age-Specific Rates, %		
Morbidity Condition	Community	1-4 Years	5-8 Years	9-12 Years
"Any Lower Respiratory Disease"	Charlotte	27.8	16.4	12.7
	Birmingham	24.0	20.6	15.9
Croup	Charlotte	10.8	7.0	4.7
	Birmingham	8.6	7.9	5.9
Bronchitis	Charlotte	13.7	7.8	6.3
	Birmingham	10.6	7.9	6.9
Pneumonia	Charlotte	14.0	8.3	8.9
	Birmingham	15.1	13.7	10.2
Hospitalization	Charlotte	4.0	1.8	1.3
	Birmingham	5.7	2.9	2.5

*Direct adjusted for sex and education of head of household. Because age distributions within the 1-4 year old age groups were similar in both communities, these rates were not adjusted for differences in the number of years at risk among 1 to 3 year olds.

TABLE 8. FOUR YEAR REPORTED RATES OF TWO OR MORE EPISODES OF EACH MORBIDITY CONDITION AMONG BLACK CHILDREN, BY COMMUNITY

Morbidity Condition	Community	Direct Adjusted* Age-Specific Rates, % 1-4 Years	5-8 Years	9-12 Years
"Any Lower Respiratory Disease"	Charlotte	15.9	7.3	7.4
	Birmingham	12.3	11.6	7.9
Croup	Charlotte	4.8	1.1	1.2
	Birmingham	2.1	3.0	2.4
Bronchitis	Charlotte	6.2	2.5	2.2
	Birmingham	3.8	3.0	2.4
Pneumonia	Charlotte	6.1	3.0	3.4
	Birmingham	4.5	5.6	4.6
Hospitalization	Charlotte	0.3	0.3	0.4
	Birmingham	0.6	0.5	0.1

*Direct adjusted for sex and education of head of household. Because age distributions within the 1-4 year old age groups were similar in both communities, these rates were not adjusted for differences in the number of years at risk among 1 to 3 year olds.

TABLE 9. FOUR YEAR REPORTED RATES OF ONE OR MORE EPISODES OF EACH MORBIDITY CONDITION AMONG WHITE CHILDREN, BY COMMUNITY EXPOSURE

Morbidity Condition	Community	Direct Adjusted* Age-Specific Rates, %		
		1-4 Years	5-8 Years	9-12 Years
"Any Lower Respiratory Disease"	Charlotte	35.0	29.9	22.0
	Birmingham	38.9	36.3	26.4
Croup	Charlotte	17.5	14.2	9.3
	Birmingham	16.3	16.3	12.2
Bronchitis	Charlotte	23.1	20.4	14.9
	Birmingham	28.7	26.2	18.6
Pneumonia	Charlotte	9.0	6.3	5.2
	Birmingham	10.3	8.5	6.3
Hospitalization	Charlotte	5.5	2.7	0.9
	Birmingham	5.8	6.1	2.4

*Direct adjusted for sex and education of head of household. Because age distributions within the 1-4 year old age groups were similar in both communities, these rates were not adjusted for differences in the number of years at risk among 1 to 3 year olds.

TABLE 10. FOUR YEAR REPORTED RATES OF TWO OR MORE EPISODES OF EACH MORBIDITY CONDITION AMONG WHITE CHILDREN, BY COMMUNITY EXPOSURE

		Direct Adjusted* Age-Specific Rates, %		
Morbidity Condition	Community	1-4 Years	5-8 Years	9-12 Years
"Any Lower Respiratory Disease"	Charlotte	19.0	17.9	11.8
	Birmingham	23.5	22.3	16.5
Croup	Charlotte	7.1	6.4	3.8
	Birmingham	4.3	8.9	7.4
Bronchitis	Charlotte	8.8	10.1	6.7
	Birmingham	16.4	14.5	12.6
Pneumonia	Charlotte	2.4	2.1	1.5
	Birmingham	2.8	2.8	2.3
Hospitalization	Charlotte	1.0	0.2	0.5
	Birmingham	0.6	1.5	0.2

*Direct adjusted for sex and education of head of household. Because age distributions within the 1-4 year old age groups were similar in both communities, these rates were not adjusted for differences in the number of years at risk among 1 to 3 year olds.

SECTION 5

EXPERIMENTAL PROCEDURES

Monitoring Human Exposure to Air Pollutants

Communities were selected within the metropolitan areas of both Birmingham and Charlotte to document exposure within each of three study sectors in each city. Within each study sector, monitoring stations were located within 1 1/2 to 2 miles of the study population. This study was part of the U. S. Environmental Protection Agency's CHESS (Community Health and Environmental Surveillance) Program. CHESS monitoring began in Birmingham in November 1969 and in Charlotte in February 1970. In each of the three study sectors selected in each city, monitoring sites were chosen to be most representative of the immediate area in which the study population resided. Each monitoring station was positioned in an area free of major obstructions to air flow and removed from the biasing effect of point sources insofar as possible. Except for Sector III in Charlotte (station located on a flat roof 16 feet above ground level) air sample inlets were positioned at head level (6 feet) to further represent respirable exposure.

Topography, prevailing weather patterns, and local or point pollutant sources determine the representativeness of any single sampling site as an indicator of air pollution exposure for a given area surrounding it. CHESS monitoring sites were placed in relatively flat areas free from point sources of pollution, e.g., a cement factory. Hence they are representative, as intended, of the air pollution exposure within a 1 1/2 to 2 mile radius of their location. The assumption is made that the study subjects spent most of their time within this radius, which is a reasonable assumption for children through age twelve (the assumption is less reasonable for children not attending neighborhood schools or for adults who may be more likely to travel out of the neighborhood during the day). Historical exposure estimates for both cities were usually based on more than one station from 1964 onward and provide a reasonably integrated estimate of long-term pollution exposure for residents of each city.

Location and Description of Monitoring Sites

The location of CHESS monitoring stations are given in Figure 1 for Birmingham and Figure 2 for Charlotte. These sites are prefaced by "C" to distinguish them from other sites which existed prior to CHESS monitoring but which were used for historical exposure estimates. National Air Sampling Network (NASN) sites are prefaced with "N", Jefferson County stations with "J", and Mecklenburg County stations with "M" to identify the respective Birmingham and Charlotte sites.

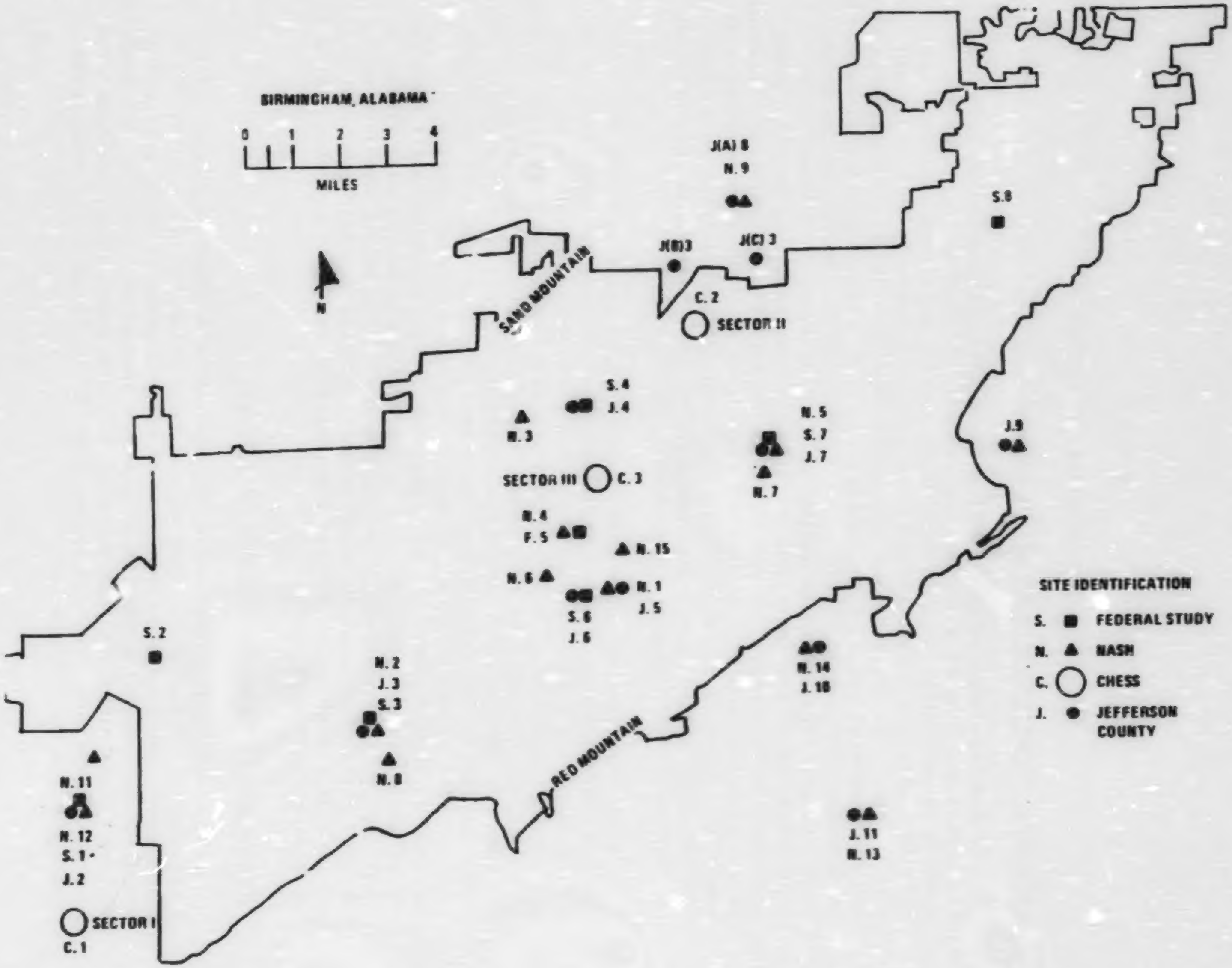
BIRMINGHAM, ALABAMA
0 1 2 3 4
MILES
N
SAND MOUNTAIN
RED MOUNTAIN
J(A) 8
N. 9
J(B)3
J(C) 3
C. 2
SECTOR II
S. 8
S. 4
J. 4
N. 3
N. 5
S. 7
J. 7
N. 7
J. 9
SECTOR III
C. 3
N. 4
F. 5
N. 15
N. 6
N. 1
J. 5
S. 6
J. 6
S. 2
N. 2
J. 3
S. 3
N. 8
N. 14
J. 10
N. 11
N. 12
S. 1
J. 2
SECTOR I
C. 1
J. 11
N. 13
SITE IDENTIFICATION
S. ■ FEDERAL STUDY
N. ▲ NASH
C. ○ CHESS
J. ● JEFFERSON COUNTY

Figure 1. Birmingham, Alabama: location of air monitoring stations.

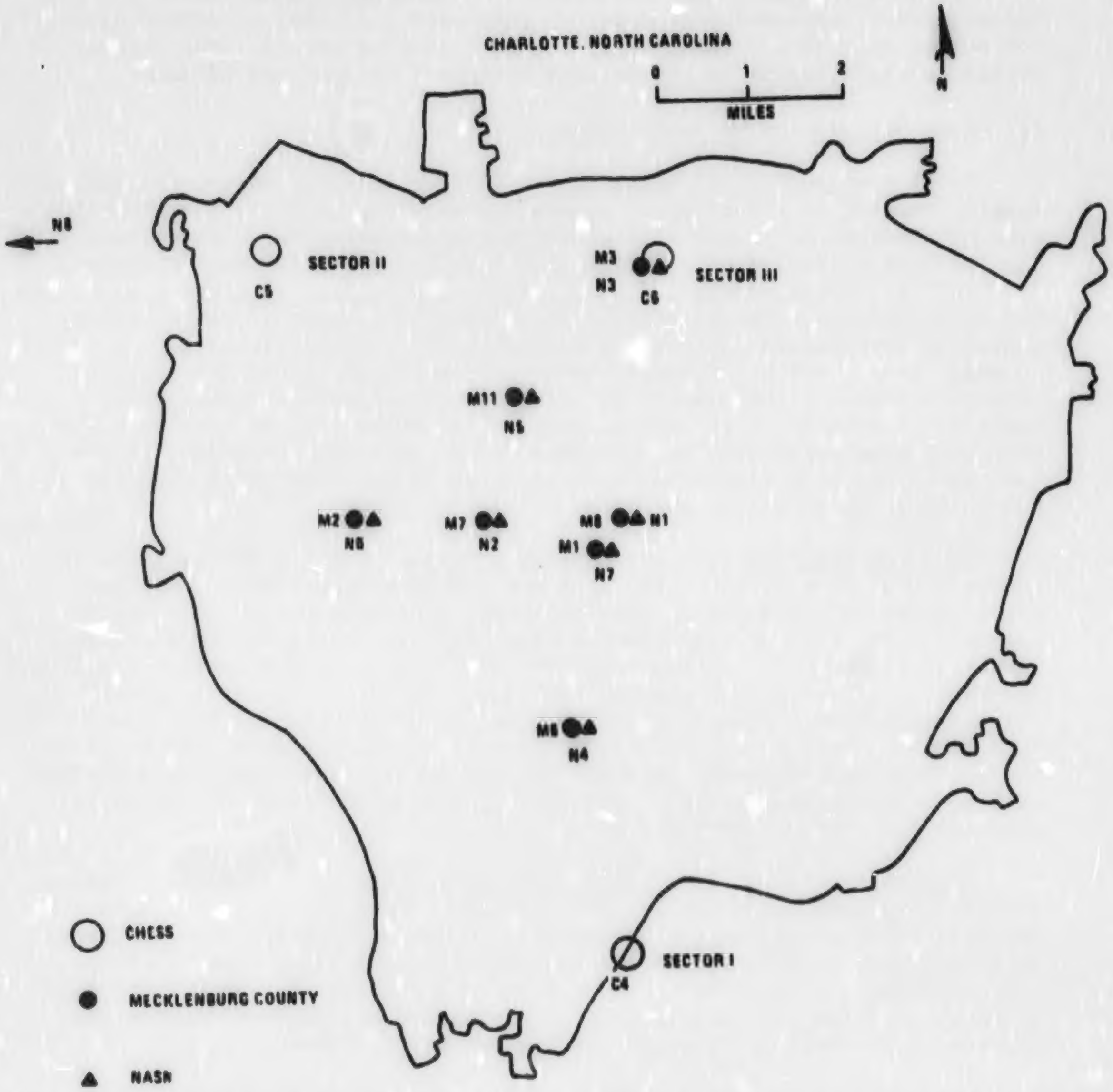
CHARLOTTE, NORTH CAROLINA
0
1
2
MILES
N
N8
SECTOR II
C5
M3
N3
C6
SECTOR III
M11
N5
M2
N6
M7
N2
M8
N1
M1
N7
M6
N4
CHESS
MECKLENBURG COUNTY
NASN
SECTOR I
C4

Figure 2. Charlotte, North Carolina: location of air monitoring stations.

CHESS measurements for respirable suspended particulate (fine particulates), total suspended particulates, suspended sulfates, suspended nitrates, and sulfur dioxide are taken daily for each 24-hour period. The 24-hour period normally begins in the morning between 8:00 a.m. and 12 noon.

Pollutant Measurement Methods (CHESS)

Total Suspended Particulate Matter (TSP) - The high volume (hi vol) air sampler mounted in a shelter of standard design was employed for the collection of atmospheric particulate matter for gravimetric and chemical analysis. Samples were collected on a 20.3 X 25.4 cm glass fiber filter. At an air flow rate of approximately 1.7 m^3 per minute, particles ranging in sizes from 0.1 to 90 microns were collected. The high volume assembly was routinely inspected, overhauled, and calibrated (with its attached rotometer) every 25 calendar days. Weight of particulate was determined by the EPA standard reference method. The results are given in micrograms per cubic meter ($\mu g/m^3$). A portion of the particulate matter collected was used to determine suspended sulfate using turbidimetric methods and suspended nitrate was determined by a reduction diazo coupling reaction and detecting the color change by automated analysis.

Fine Particulates (Cyclone Separator Method) - For estimating the concentration of fine particulates that correspond to respirable suspended particulates (RSP), a small stainless steel cyclone, 1.27 cm in diameter, was used. The cyclone operation is such that two fractions are produced: The heavier, larger particles fall into a plastic cup, the smaller respirable fraction is drawn up into the air vortex and impinges on a 37 mm glass fiber filter mounted in a plastic cassette between the cyclone and the vacuum pump. In a similar fashion, an open-faced cassette is used in parallel to collect the total suspended particulate (TSP). The cyclone and open-face cassette each has a critical orifice placed between the vacuum pump and the filter cassette for sample flow control.

Fine Particulates (Five Stage Cascade Impactor) - A five-stage cascade impactor was used for detailed particulate monitoring. Particles were separated from the airstream by inertial impaction on glass fiber "filters" into five aerodynamically sized fractions: above 5.50 microns, 2.40 to 5.50 microns, 1.75 to 2.40 microns, 0.93 to 1.75 microns, and 0.01 to 0.93 microns. The impactor was mounted in a standard high volume shelter and operated at a sampling flow rate of 566 liters per minute.

Suspended Sulfate (SS) - Twenty-four suspended sulfate measurements were made from CHESS high volume particulate samples. A 1.9 cm x 20.3 cm strip of the exposed high volume filter was refluxed and the sulfate ion concentrations determined by spectrometric methods.

Suspended Nitrate (SN) - Twenty-four hour suspended nitrate measurements were made from CHESS high volume particulate samples using a strip of the exposed filter and spectrometric methods similar to the sulfate analysis above.

Sulfur Dioxide (SO_2) - Twenty-four SO_2 measurements were made according to the West-Gaeke reference method as published in the Federal Register with minor changes in air flow rate and absorbing solution volume.

NASN Data - The CHESS program supplements its data collection with data from the National Air Sampling Network (NASN) whenever possible.

Precision of Measurements

The CHESS program conducted tests to determine the precision of environmental measurements. These tests were done in Birmingham, Charlotte and Greensboro, North Carolina. Duplicate sensors were installed at air monitoring sites within these cities on a daily basis for 8 months in Birmingham, and for 6 months in Charlotte. The comparison of the regular samples to duplicates was used to determine statistically the precision of CHESS measurements for TSP and SO_2. The arithmetic mean errors for TSP and SO_2, were 6.1% and 27.1% respectively.

Quality Control

An effective quality control program is an essential element of any environmental surveillance system, since the data outputs of an air sampling program are subject to any sources of error. Effective, real time controls are essential to minimize field errors, systematic drift, chemical laboratory errors, data transfer errors, computer punch card errors, analysis errors, etc. A quality control program was used for the CHESS data in this study, but not for the earlier data.

Long Term Exposure Trends

Because CHESS monitoring was designed to be coupled with epidemiological health studies and because CHESS sites were specifically selected to represent the study area, CHESS data were used as the standard for this report. Equivalency adjustment of non-CHESS data obtained from different locations used in prior years has been made only to present an appropriately scaled trend of past exposure. In many instances historical data were unavailable for some pollutants of interet, or were available in different forms, or represented an area outside the CHESS study area. The annual standard for TSP is a geometric mean rather than an arithmetic mean like those for sulfur dioxide and nitrogen dioxide. This decision was based on the distribution of TSP values and not health data. Since virtually all ambient pollutant concentrations are positively skewed, the use of a geometric mean only for TSP is inconsistent. Certainly, the use of a geometric mean to estimate human exposure, minimizes differences in peak episodic exposures between communities. Nevertheless, this is the reason for the use of a geometric mean of TSP as well as an arithmetic mean in this report.

Relationships between RSP and TSP were analyzed using current data and have been applied to historical data. On the basis of the correlations presented, this approach provided reasonable estimates for specific pollutants for the years during which measured data were not available. Since respirable suspended particulates were not collected routinely prior to CHESS studies, all RSP values prior to 1969 are estimates based on collections of TSP. Because of the special complexities of historically estimating RSP exposure and the resulting uncertainties these estimates are presented here, but not in the body of the report. Although measured RSP data are not available prior to 1969, it is possible to estimate RSP directly from TSP under certain conditions which are discussed fully in the report of Hinton, et al.

Comparisons of measured data from cyclone RSP and high volume TSP were made for both Birmingham and Charlotte during the period in which the cascade impactor was being evaluated. It was found that as TSP concentrations increase, RSP concentrations do not increase proportionately but rather at constantly reducing proportions of the total, i.e. the relationship of RSP to TSP was that of a monotonically increasing function with decreasing slope.

Graphical analysis of the concentrations within each size range as a function of relative humidity showed that while the slopes vary, concentrations decrease with increased humidity. It is postulated that this is due primarily to the aerodynamic separation technique. As explained previously, the highly humid particle would be collected as though it were a larger particle with a smaller percentage reaching either the filter or the human lung. (Although laboratory equilibration of the filter may remove a portion of the moisture further reducing the weighed mass, this effect would apply equally to both RSP and TSP.) This partial explanation indicates the need for further study. Other factors such as either vertical or horizontal wind dispersion, and the composition of total particulates would slightly affect the ratio of RSP to TSP.

Cyclone RSP is the monitoring method for the respirable fraction upon which the CHESS study was based. Impactor data are available for only a short period during the CHESS study; therefore, cyclone RSP to hi vol TSP ratios were used to provide a basis for estimating prior RSP concentrations from hi vol TSP exposure.

Within the accuracies of past high volume measurements, reasonable estimates of past RSP exposure may be obtained by multiplying the TSP value by the following percentage factors:

	TSP Range in $\mu g/m^3$	Percentage RSP
Birmingham	80-100	59.0
	100-120	46.0
	120-140	43.0
	140-160	37.5
Charlotte	60-80	55.0
	80-100	47.5

Estimates of RSP exposure obtained in this manner are given in Tables 11 and 12 for 1960 through 1971.

Simultaneous collections of data from the cascade impactor sampler, the RSP cyclone and the high volume sampler were analyzed and it was found that when the appropriate percentage RSP was applied to the TSP data of the hi vol, the resultant value obtained was between the impactor value and the cyclone value, thus lending further credance to the above percentages. Because of the complexity of pollution sources in both cities, a predictive approach was considered invalid for estimating pollutant values for years in which measurements for that pollutant were not available.

To develop estimates of past exposure, before the CHESS stations were installed, available local, state and federal data were obtained (primarily for the years 1968 through 1972). These data were taken by several different agencies, under different conditions and for different purposes. Annual averages were available as arithmetic means, and/or geometric means in some cases, and in frequency distributions (without means) in others. The number of samples per year ranged from less than 20 to a maximum of 26 for years when samples were taken at two-week intervals. Equipment malfunction and other data losses were potential sources of bias. This bias was minimized by comparing valid quarterly averages to corresponding quarters in other years and adjusting the annual average where indicated.

Prior to 1964, only one station in each city was in operation and sampled one day every two weeks. To approximate annual exposure, geometric means were used supplemented by 50th percentiles when geometric means were not available.

Annual geometric means from county data were also used for the more recent years. A trend line for particulates drawn on the basis of recent data indicates higher past exposure for Birmingham (Figure 3) than one based soley upon the values of one station. TSP exposure in Birmingham prior to 1964 is represented by the solid line in Figure 3 and represents a "best-judgment" decision based upon the relative accuracy and validity of both sets of data. Values are given in Table 11. This trend is in general agreement with a NASN single station analysis for the years 1958-1964.

A similar approach was used on the Charlotte particulate data. Independent analysis of both recent and past exposure data in this case, however, resulted in almost identical trend lines (Figure 4). Exposure values are given in Table 12. As shown in Figures 5 and 6, CHESS sector RSP values for Birmingham vary from 13 to 36 percent lower than the historical line calculated from the TSP values, while those for Charlotte are 6 to 30 percent lower. Less data were available for other pollutants. These data are summarized in Tables 11 and 12 and displayed for sulfur dioxide and suspended sulfates in Figures 7 through 10. Complete historical estimates for TSP by sector are summarized in Tables 13 and 14. (Further details and the original figures may be found in "Human Exposure to Fine Particulates in the Southeastern CHESS Area: Birmingham, Alabama and Charlotte, North Carolina," Final Draft, November 1974 by David O. Hinton, et al., Human Studies Laboratory, EPA-NERC, Research Triangle Park, North Carolina 27711 This report is available upon request and will be published in a forthcoming EPA Particulate Monograph.)

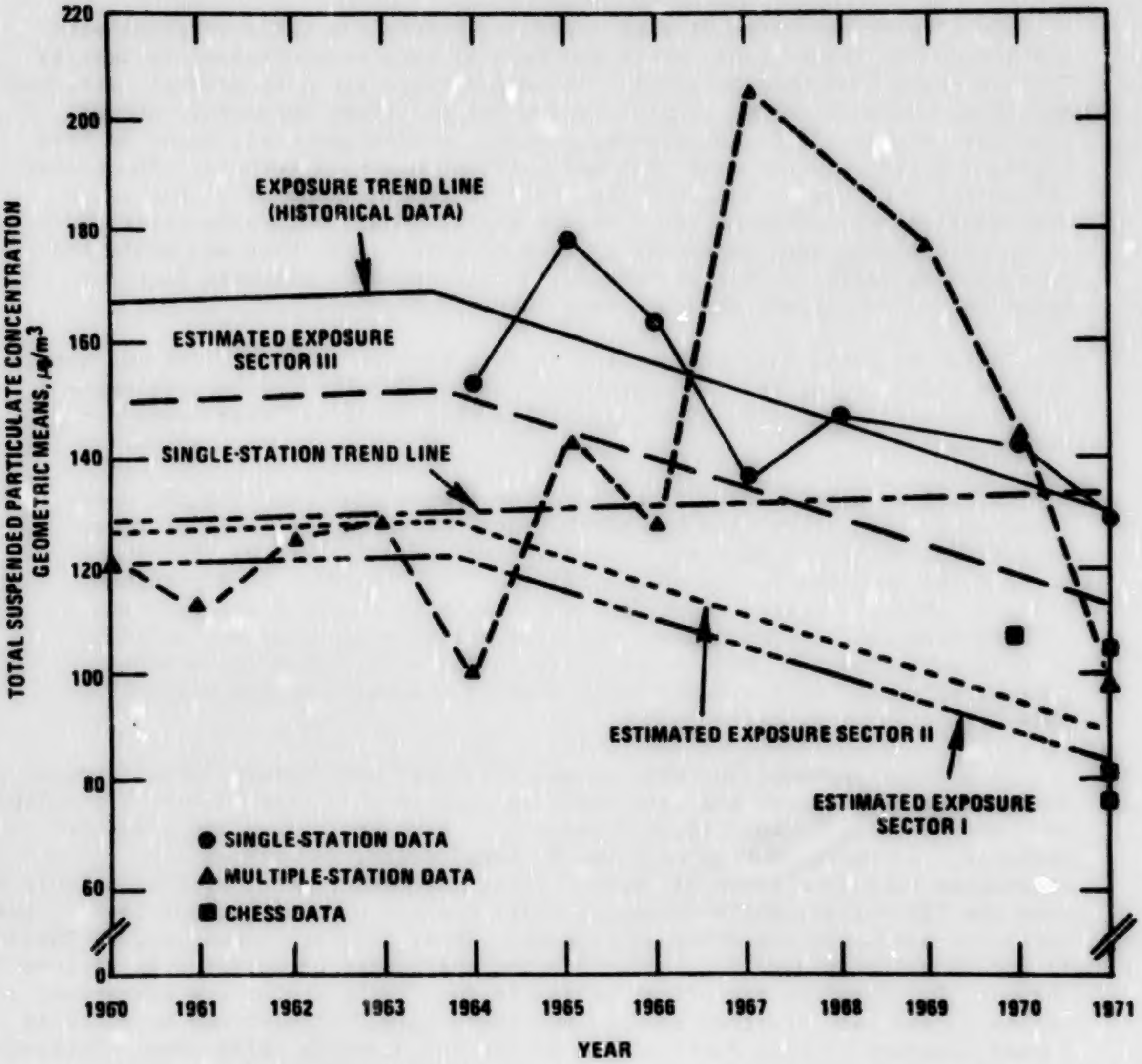

Figure 3. Birmingham, Alabama: total suspended particulate matter, historical exposure.

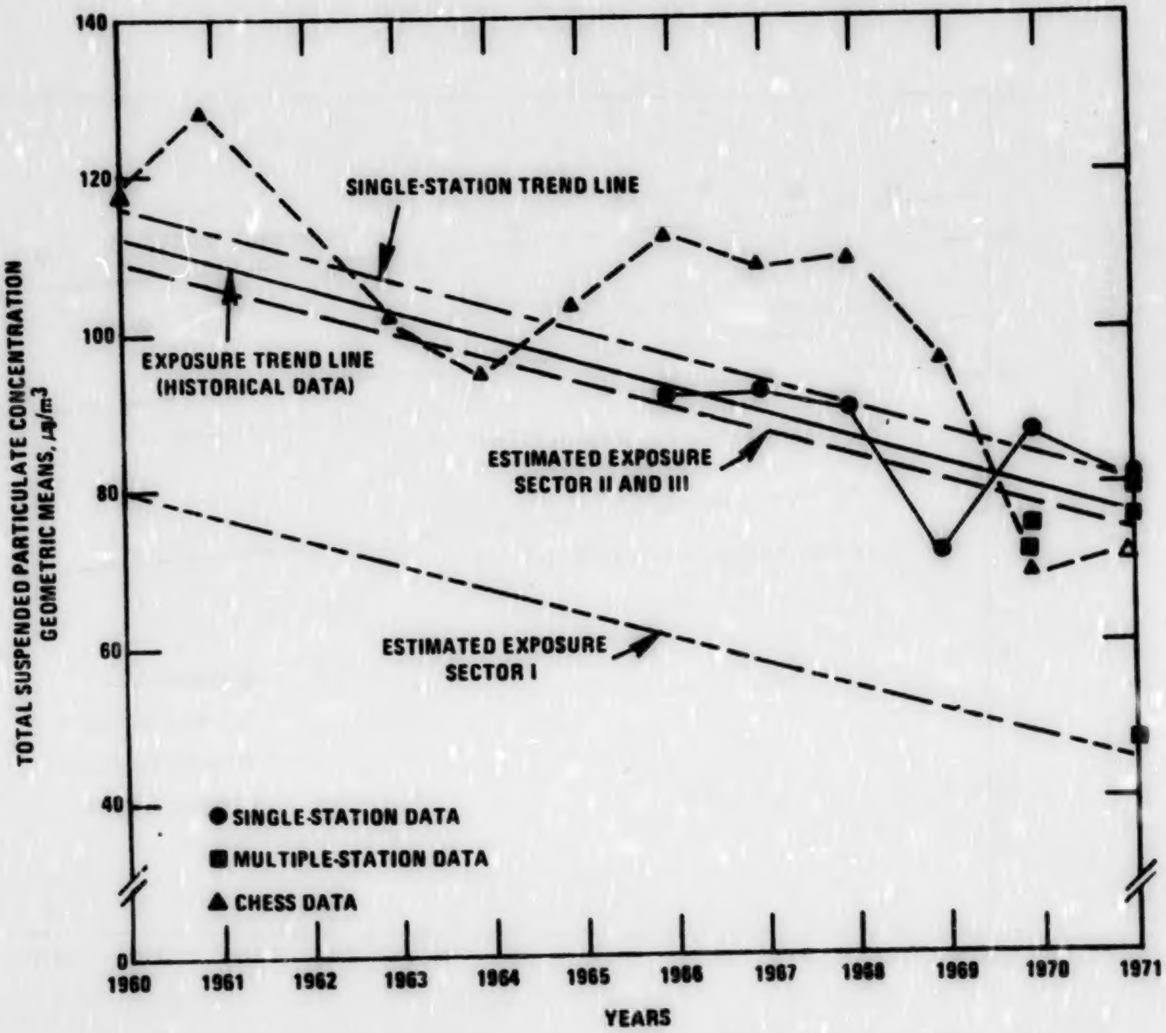

Figure 4. Charlotte, North Carolina: total suspended particulate matter, historical exposure.

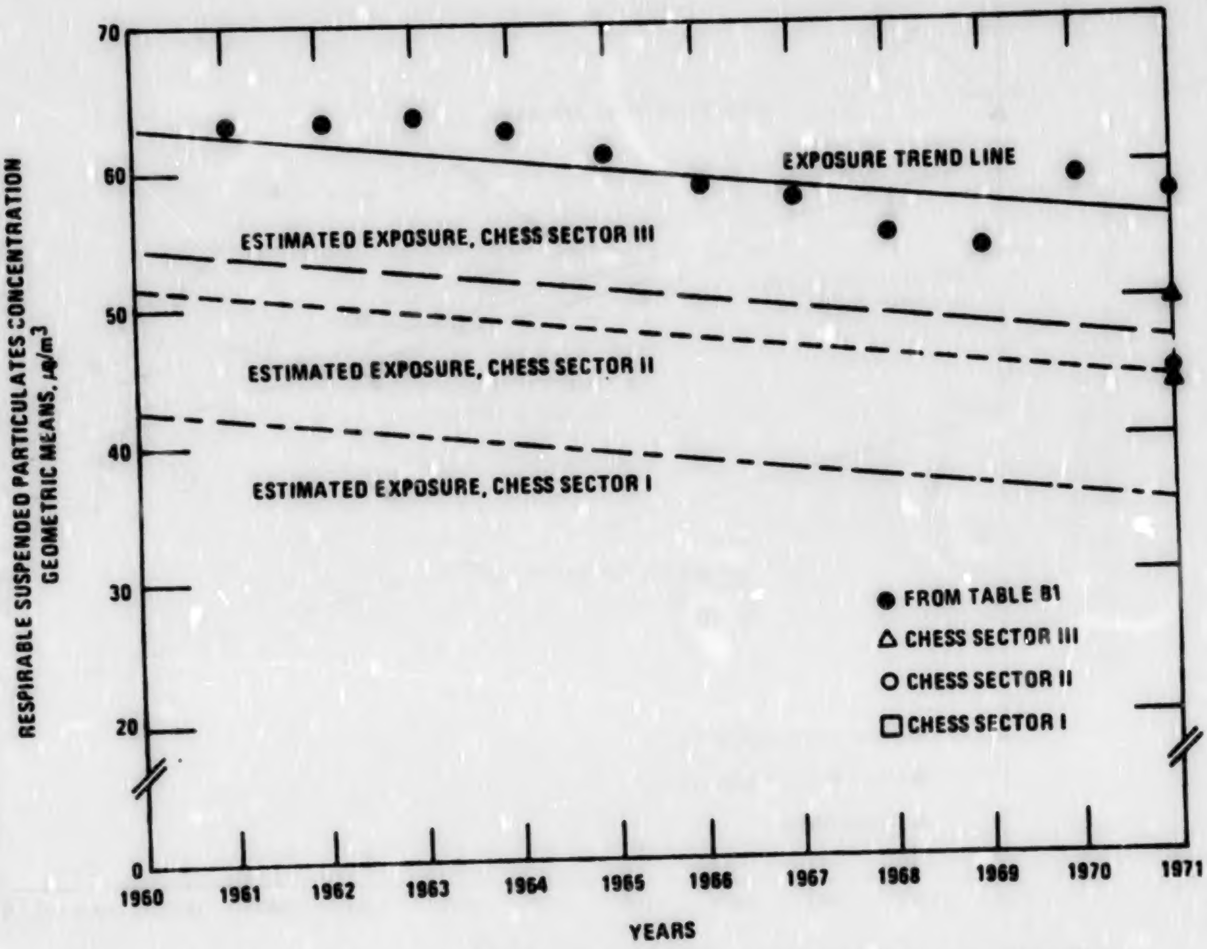

Figure 5. Birmingham, Alabama: respirable suspended particulates, historical exposure.

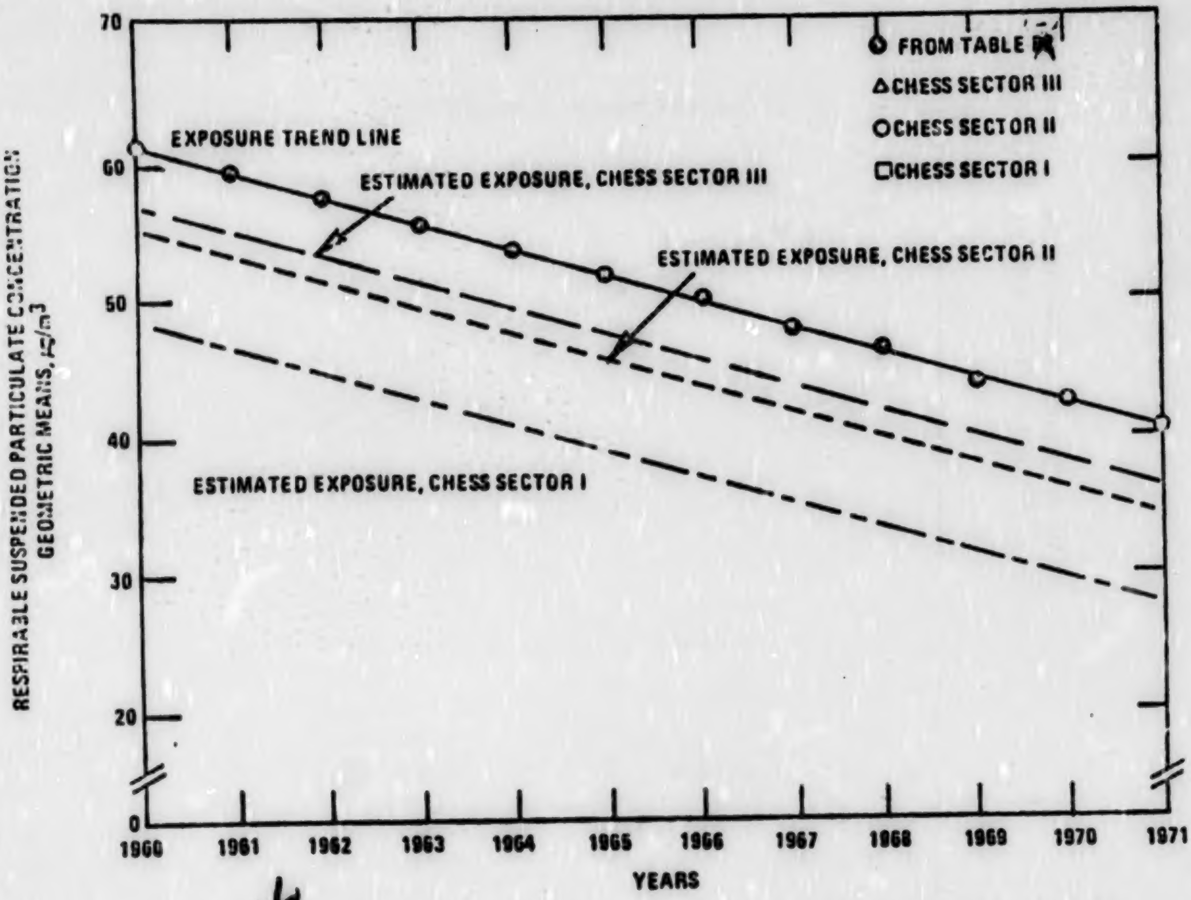

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Figure [illegible]. Charlotte, North Carolina: Respirable suspended particulates, historical exposure.

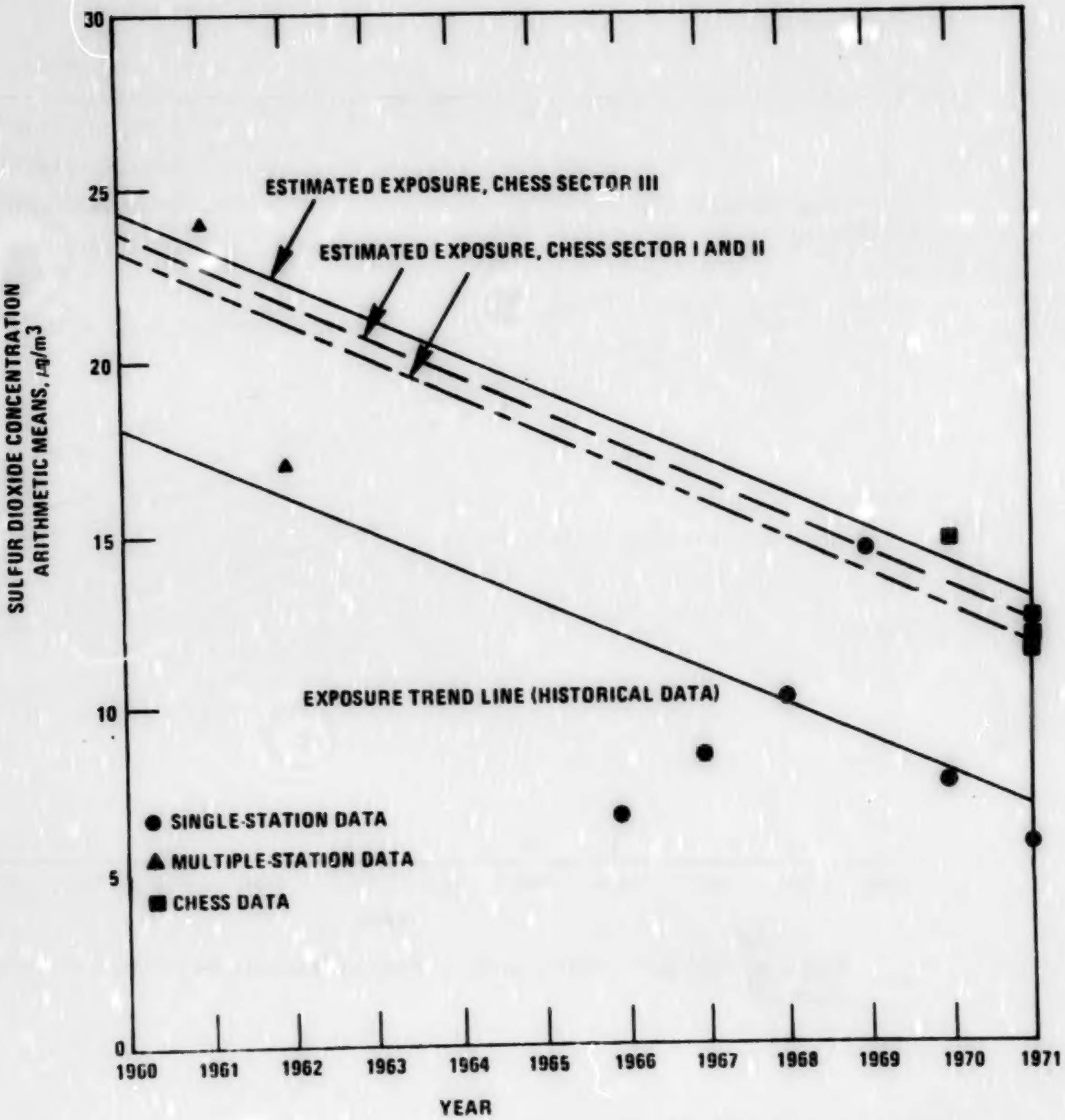

Figure 7. Birmingham, Alabama: sulfur dioxide, historical exposure.

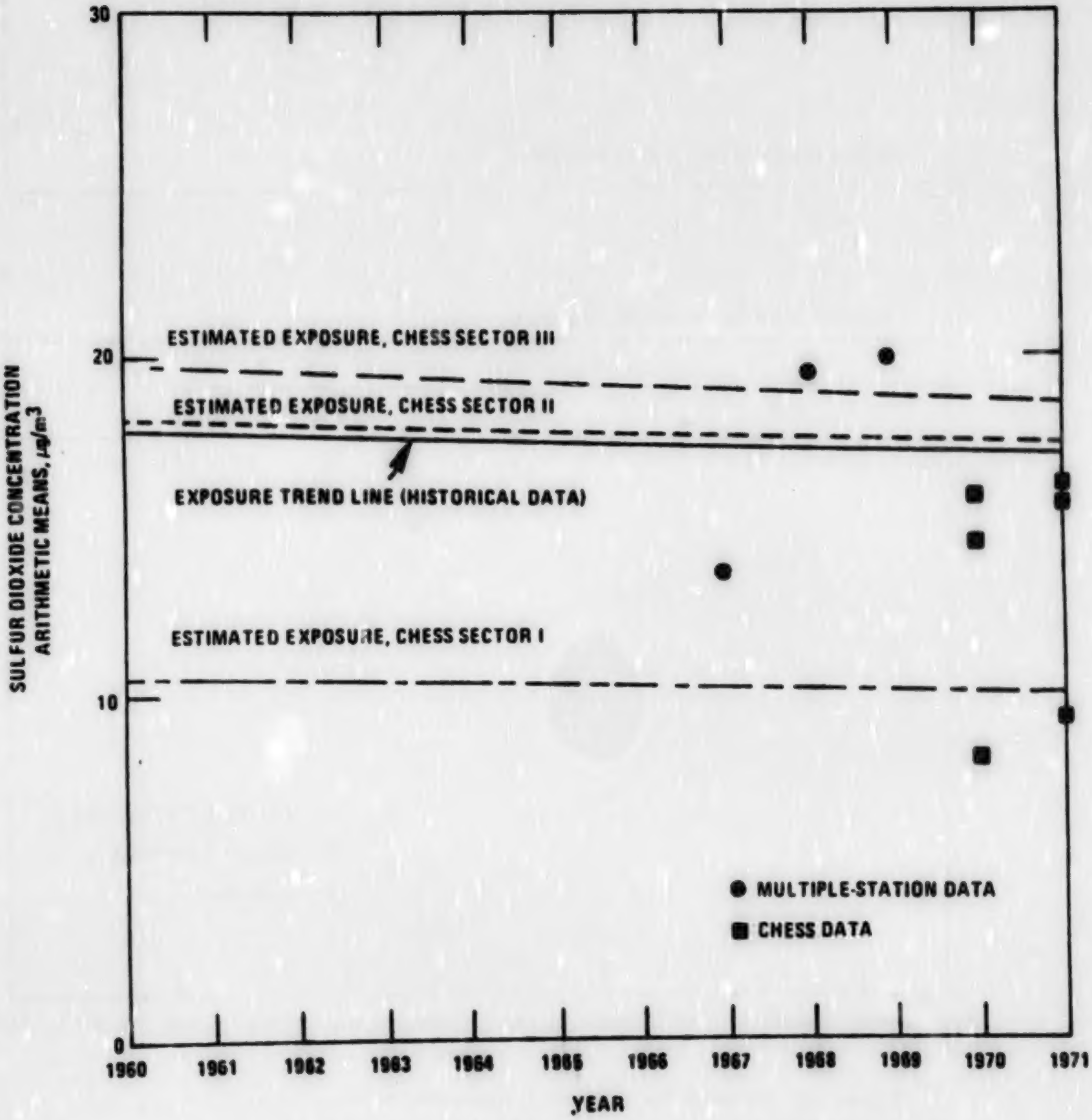

Figure 8. Charlotte, North Carolina: sulfur dioxide, historical exposure.

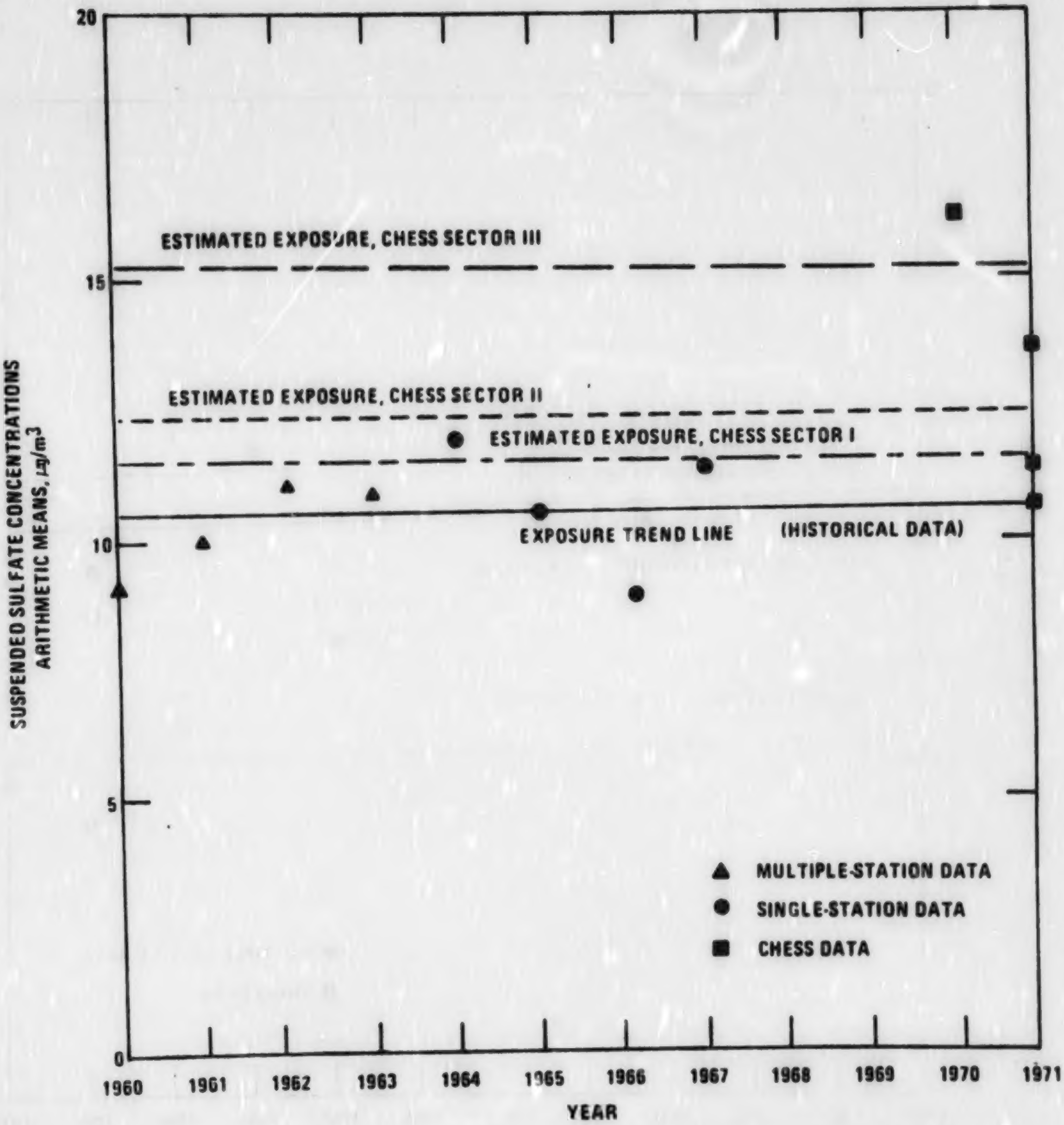

Figure 9. Birmingham, Alabama: suspended sulfates, historical exposure.

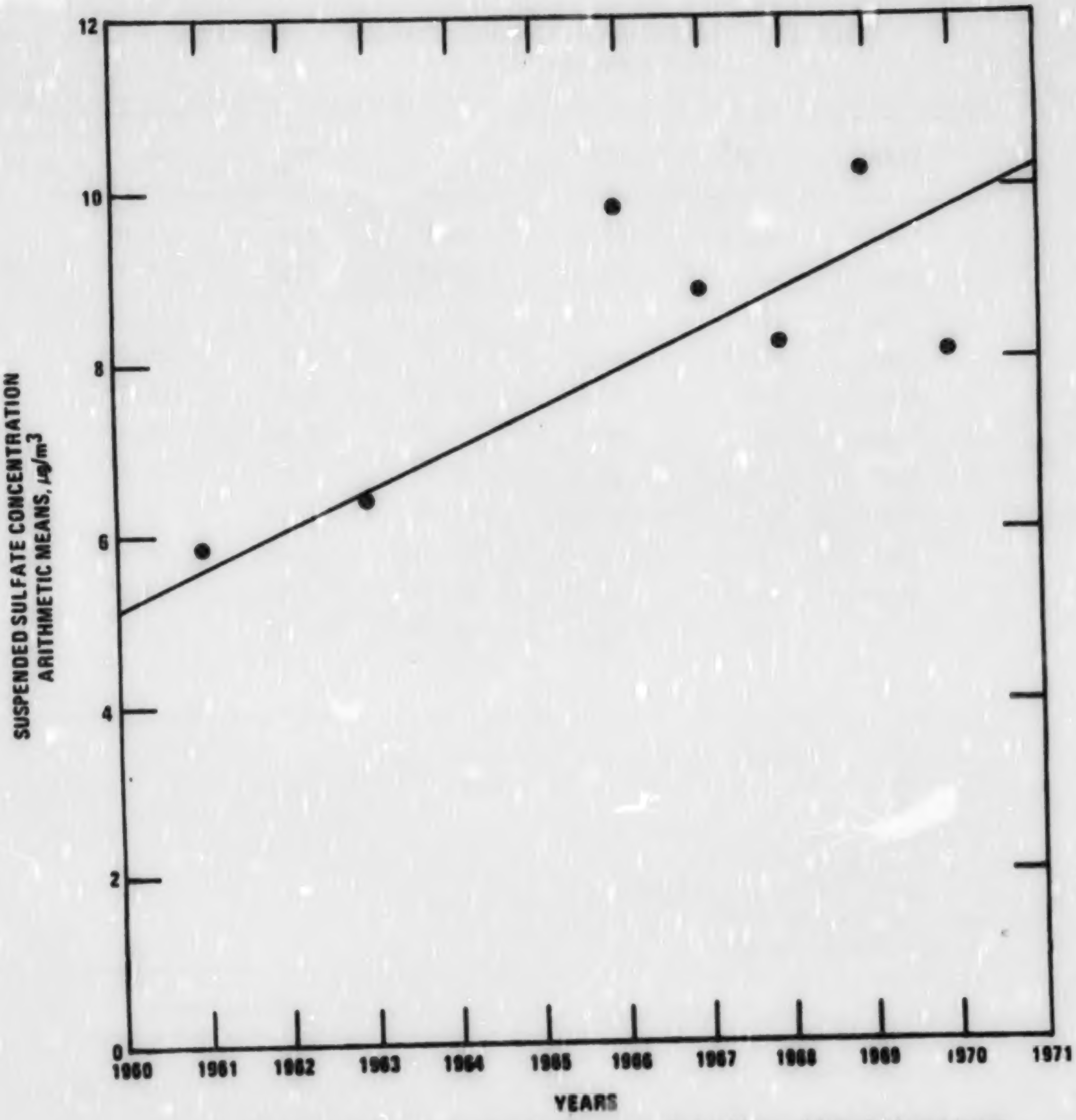

Figure 10. Charlotte, North Carolina: suspended sulfates, historical exposure.

TABLE 11. BIRMINGHAM HISTORICAL EXPOSURE (1960-1971)
(Yearly Averages)**

YEAR	TSP[a]	RSP[b]	SO_x	NO_x	SO_2
1960	166.5	62.4	9.2^{c}	2.2^{c}	<25.0*
1961	167	62.6	10.0^{c}	2.1^{c}	<25.0*
1962	168	63.0	11.1^{c}	2.1^{c}	<25.0*
1963	168.5	63.2	10.9^{c}	2.1^{c}	<25.0*
1964	166	62.3	11.9^{g}	2.4^{c}	<25.0*
1965	161.5	60.6	10.5^{g}	2.8^{c}	5.5^{f}
1966	155.5	58.3	8.7^{g}	3.0^{c}	6.8^{f}
1967	153	57.2	11.4^{f}	3.0*	9.5^{f}
1968	145.5	54.6	10.7*	3.0*	10.7*
1969	142.5	53.4	10.7*	3.1*	14.6^{c}
1970	136	58.5	16.0^{c}	2.0^{c}	14.9^{c}
1971	133	57.2	11.8^{d}	2.5^{d}	12.1^{d}

a - Values obtained from trend line Figure 3
b - TSP x 0.375 (thru 1968). TSP x 0.43 (1969-71).
c - One site only
d - CHESS data
e - NASN data
f - Jefferson County data
g - Federal study data
* - Estimated value

**Twenty-four hour integrated estimates of concentrations were measured, expressed in micrograms per cubic meter. For each year the average of all daily estimates was completed.

TABLE 12. CHARLOTTE HISTORICAL EXPOSURE (1960-1971)
(Yearly Averages)**

YEAR	TSP[a]	RSP[b]	SO_x	NO_x	SO_2
1960	112	61.6	4.3[c]	1.6[c]	17.0*
1961	109	59.9	5.8[c]	2.1[c]	16.9*
1962	105	57.8	6.1*	2.0*	16.8*
1963	102	56.1	6.4[c]	1.9[c]	16.7*
1964	98	53.9	7.5*	2.2*	16.6*
1965	94.5	51.9	8.6*	2.3*	16.5*
1966	91.5	50.3	9.8[c]	2.5[c]	16.4*
1967	87.5	48.1	8.8[c]	1.8[c]	13.6[f]
1968	84.5	46.5	8.2[c]	1.6[c]	19.4[f]
1969	80	44.0	10.2[c]	1.3[c]	19.9[f]
1970	77	42.4	10.4[d,e]	0.7[d]	12.8[d]
1971	74	40.7	9.6[d]	1.7[d]	16.3[d,f]

a - Values obtained from trend line Figure 4.
b - TSP x 0.55
c - One site only
d - CHESS data
e - NASN data
f - Mecklenburg County data
* - Estimated value

**Twenty-four hour integrated estimates of concentrations were measured, expressed in micrograms per cubic meter. For each year, the average of all daily estimates was completed.

TABLE 13. TOTAL SUSPENDED PARTICULATES CHESS EQUIVALENT EXPOSURE BIRMINGHAM, ALABAMA

Year	TSP History	Sector I	Sector II	Sector III	Sector Average
1960	166.5	122.0	125.0	148.5	131.8
1961	167.0	122.5	126.0	149.0	132.5
1962	168.0	123.0	127.0	150.0	133.3
1963	168.5	123.0	128.0	151.0	134.0
1964	166.0	120.0	126.0	149.5	395.5
1965	161.5	115.0	123.5	145.0	127.8
1966	155.5	110.0	117.5	140.0	122.5
1967	153.0	105.0	111.5	135.0	117.1
1968	145.5	100.0	105.5	129.0	111.5
1969	142.5	94.5	100.0	124.0	106.1
1970	136.0	88.5	94.0	120.0	100.8
1971	133.0	84.5	88.0	114.5	95.6

TABLE 14. TOTAL SUSPENDED PARTICULATES CHESS EQUIVALENT HISTORICAL EXPOSURE CHARLOTTE, NORTH CAROLINA

Year	TSP History	Sector I	Sector II	Sector III	Sector Average
1960	112.0	80.0	108.0	108.0	98.6
1961	109.0	76.5	105.0	105.0	95.5
1962	105.0	73.5	102.0	102.0	92.5
1963	102.0	69.5	98.5	98.5	88.8
1964	98.0	65.5	95.0	95.0	85.1
1965	94.5	63.5	90.5	90.5	81.5
1966	91.5	60.0	87.0	87.0	78.0
1967	87.5	56.5	84.0	84.0	74.8
1968	84.5	53.5	82.0	82.0	72.5
1969	80.0	50.0	78.5	78.5	69.0
1970	77.0	48.0	75.0	75.0	66.0
1971	74.0	43.5	71.5	71.5	62.1

SECTION 6

RESULTS AND DISCUSSION

Environmental Exposure

Estimates of ambient air pollutant exposure levels for the entire lives of the oldest study children are summarized in Table 15. Youngest children, aged 1 to 4 years, were exposed to pollutant levels only from 1968 through 1971. Children aged 12, on the other hand, were exposed to ambient air pollutant levels as early as 1960. Particulate levels decreased from 1960 through 1971 in both communities. Estimates suggested that annual total suspended particulates ranged from 133 to 169 $\mu g/m^3$ in Birmingham, and from 74 to 112 $\mu g/m^3$ in Charlotte for the twelve years preceding the study. These values confirmed the expected exposure gradient.

Annual sulfur dioxide levels were less than 25 $\mu g/m^3$ in both communities. Suspended sulfate levels in Birmingham (9 to 16 $\mu g/m^3$) were higher than those in Charlotte (4 to 10 $\mu g/m^3$). An important observation is that such suspended sulfate levels can occur in the presence of low sulfur dioxide levels. Annual suspended nitrate levels were low in both communities. Thus, in comparison to Charlotte, residents of Birmingham were exposed to elevated levels of particulate matter in the presence of low levels of sulfur dioxide. Furthermore, these exposure differences appeared to be relatively constant for the lifetime of even the oldest group of children in the study.

Questionnaire Response

Response rates were excellent in both communities, although significantly fewer families returned the questionnaires in Charlotte (Table 16). In Birmingham, there was no difference in return rates by race; in Charlotte, the return rates were significantly lower for blacks, but the absolute difference was small (84 vs. 89%). For those questionnaires which were returned, 90 to 94% were completed for all city-race categories. Only a small proportion of children had to be excluded from the analysis because of missing information (Table 17a, 17b). Missing information about a child's lower respiratory disease morbidity was the major reason for exclusion. It was not possible to determine return rates among families by the presence or absence of one or more children with a history of asthma because asthma prevalence was not determined among the nonrespondents.

Family Characteristics

In both communities, 87 to 95% of the families had resided there for three or more years (Table 18a, 18b). Educational attainment of fathers was highest among younger persons and whites. Black parents in Birmingham smoked somewhat less than the other three parental groups, which were quite comparable. The median age of female parents or guardians ranged from 34 to 37 years. Blacks in both communities had a higher proportion of mothers or female guardians, aged 50 years or more, than did whites (Table 19a, 19b). When compared to blacks within each community, whites tended to have fewer persons per household, but more rooms and more air conditioning in their households (Table 19a, 19b). When communities were compared with one another within each racial group, families were generally similar in regard to these attributes. Families with one or more children with a history of asthma were comparable to families with children without a history of asthma.

Prevalence of Asthma in Families

A total of about 75 black and 150 white children were reported with some history of asthma in each community (Table 21). The age distribution of white asthmatic children was comparable to white nonasthmatic children, but among black asthmatic children, 1 to 4 year olds were less frequent than their nonasthmatic counterparts. The reasons for this are not clear. "Asthma ever diagnosed by a doctor" was more prevalent among Birmingham *white* children than among the other three groups ranging from 3 to 14% with an excess of males to females for all four city-race groups (Table 22). The prevalence of "asthma active during the past two years" for all ages ranged from 2 to 5% and also showed a male excess for both races (Table 23). Almost all of the white asthmatic children in the youngest age group were two or more years old. The proportion of active to all asthmatic children generally decreased with age for both boys and girls although it was somewhat higher in boys when compared to girls (Table 24).

The prevalence of asthma in *parents* differed from that of children (Table 25). For parents of both races, asthma prevalence was higher in Charlotte than in Birmingham. Prevalence rates ranged from 2 to 9% overall, but did not vary with age or parental education. Whereas "asthma ever diagnosed" did not differ by sex in whites, it was higher among black females than among black males in both cities. Females reported a significantly higher proportion of active asthma for all city-race groups when compared to males. Asthma prevalence in children and adults clustered in families. One or more children with asthma were reported three and eight times more frequently in families with one or both parents with asthma, respectively, when compared to families in which neither parent had asthma (Table 26). Asthma prevalence in children was also related to the activity of parental asthma, but the increases were statistically significant in Charlotte only (Table 27). Parental cigarette smoking habits, education, duration of residence, household size, and air conditioning were the same for families with and without any parents with asthma.

Lower Respiratory Illness in Children

Sex distributions were generally comparable among all children and about 15% of all children without asthma were in the 1 to 4 years of age group (Table 20). Morbidity rates, direct-adjusted for sex and education of the head of household, were tabled by four-year age intervals for blacks (Tables 7, 8) and whites (Tables 9, 10). Respiratory disease rates decreased with age among children of both races in both communities. When compared to whites, blacks reported higher rates of pneumonia and lower rates of "any lower respiratory disease," croup, bronchitis, and hospitalization. Rates for two or more episodes were lower, of course, but showed the same pattern with respect to age and race. Among blacks, the direct-adjusted rates of both one or more and two or more episodes of "any lower respiratory disease", croup, and bronchitis among 5 to 12 year olds and pneumonia among all ages were generally higher in Birmingham (Table 7, 8). Nevertheless, the rates for one or more episodes of hospitalization and two or more episodes of all conditions except "any lower respiratory disease" and one or more and two or more episodes of croup among 1 to 4 year olds were higher in Birmingham (Tables 9, 10). For whites, rates of two or more episodes of pneumonia and hospitalization were quite low.

Because of the black/white differences in family characteristics and in reported morbidity patterns, the relationship of reported morbidity to air pollution exposure, age, sex, and education of the head of household was tested statistically in a linear categorical analysis of variance model separately for each race. Chi-square and statistical significance levels for morbidity relationships in black children are summarized in Table 3. Morbidity rates generally decreased significantly with age but did not vary by sex. Morbidity reporting was significantly increased in children from families with a high school or greater education for one or more episodes of bronchitis and the increase approached significance ($0.10>p>0.05$) for two or more episodes of bronchitis. Only one or more episodes of pneumonia was significantly higher in Birmingham but significant "city" interaction terms were observed for this as well as five other of the nine morbidity by number of episode models. (Rates for two or more episodes of hospitalization were too low to be tested in the model.) Significant "city" differences were not observed for both episodes of bronchitis and two or more episodes of pneumonia, i.e., for those morbidity conditions in which significant "city" interactions did not occur in the saturated model.

Reduced statistical models were used to account for the morbidity conditions reported for black children in which one or more significant "city" interaction terms were found (These models are presented in detail in Appendix B). For one or more episodes of "any lower respiratory disease," the city within sex comparison showed that for females the rate was significantly increased in Birmingham as compared to Charlotte, but no city differences were detected among males. Rates also decreased significantly with age and the increased rate in children from better educated families when compared to less educated families approached significance ($0.10>p>0.05$). The city within sex comparisons did not differ significantly for two or more episodes of "any lower respiratory disease;" rates declined significantly with age, but only among Charlotte males, and rates were significantly increased in children from better educated families in Birmingham, but not in Charlotte.

One or more episodes of croup was significantly higher in Birmingham, when compared to Charlotte, in children from households with a high school or greater education, but the city difference was not significant for children from less educated families. One or more episodes of croup decreased significantly with age but did not vary significantly by sex. For two or more episodes of croup, the city within age comparison was significantly increased only in Birmingham among 5-8 year olds, and rates did not vary significantly with either sex or education of the head of the household. For one or more episodes of pneumonia, the city within SES comparisons detected that the Birmingham rate was significantly greater than the Charlotte rate in children from better educated families. A similar pattern was observed in children from less educated families, although the test for city differences only approached significance (0.10>p>0.05). Rates decreased significantly with age but did not vary significantly by sex. One or more episodes of hospitalization was significantly increased in Birmingham when compared to Charlotte, and these rates also did not vary significantly by sex.

In summary, for black children, when the city (pollution) effect was distinguished from effects of age, sex, and socioeconomic status in the analysis of variance, rates of one or more episodes of "any lower respiratory disease," croup, pneumonia, and hospitalization as well as two or more episodes of croup were significantly higher in Birmingham when compared to Charlotte. In no case were city differences found in which morbidity rates in Charlotte were significantly higher than those in Birmingham. When significant, morbidity rates decreased with age and were higher in children from households with a high school or greater education. No significant differences by sex were found.

Chi-square and significance levels for morbidity relationships in white children are summarized in Table 4. When compared to Charlotte, rates in Birmingham were significantly higher for one and two or more episodes of "any lower respiratory disease," two or more episodes of croup and two or more episodes of bronchitis, i.e., for those morbidity conditions in which significant "city" interactions did not occur in the saturated model. Rates decreased significantly with age for all four of the above morbidity conditions. Rates were significantly increased in males and in children from households with a high school or greater education in all conditions except for two or more episodes of croup.

As for black children, reduced statistical models were used to account for the morbidity conditions reported for white children in which one or more significant "city" interaction terms were found (These models are presented in detail in Appendix B). One or more episodes of croup was significantly increased in Birmingham when compared to Charlotte, but the rates did not vary significantly by sex. For one or more episodes of bronchitis, the city within sex comparisons indicated significantly increased rates in Birmingham males when compared to those in Charlotte. Similar results (0.10>p>0.05) were observed for females. Bronchitis rates decreased significantly with age and were significantly increased in children from better educated families when compared to children from less educated families.

One or more episodes of pneumonia was significantly increased in Birmingham when compared to Charlotte, but these rates did not vary significantly by sex. For one or more episodes of hospitalization, the city with SES comparison showed hospitalization rates were significantly increased in Birmingham as compared to Charlotte among children from better educated families, but no significant city differences were found in children from less educated families. Hospitalization rates decreased significantly with age, but did not vary significantly by sex.

In summary for white children, rates of all morbidity conditions were significantly higher in Birmingham when compared to Charlotte. For white children, as for black children, in no case were city differences found in which morbidity rates in Charlotte were significantly higher than those in Birmingham. When significant, morbidity rates decreased with age and were higher in males and in children from households with a high school or greater education.

Model adjusted rates for "city" effect in the saturated linear model for categorical data were computed. For blacks, rates were higher in Birmingham except for bronchitis (Table 28). For whites, model-adjusted morbidity rates were higher in Birmingham for all conditions with relative increases ranging from 1.07 for one or more episodes of croup (in which there was a second order interaction involving "city") to 1.64 for two or more episodes of bronchitis (Table 29). Statistically significant differences in morbidity reporting by sex and by education of the head of the household were in the same order of magnitude as those for city. For example, for one or more episodes of "any lower respiratory disease," the model adjusted (for age, city, and SES) rates were 20.5% for females and 32.8% for males and model adjusted rates for children from households with less than a high school education were 28.9% compared to 33.4% for children from households with a high school or greater education.

As expected, children with a history of asthma had higher rates of all morbidity conditions than did their nonasthmatic counterparts (Tables 30-34). This pattern was observed for one or more and two or more episodes of each condition. Children with active asthma generally had higher morbidity rates than children with asthma diagnosed but not active; exceptions to this were croup among whites in Charlotte and blacks in Birmingham, pneumonia among whites in Birmingham, and hospitalization among blacks in both cities and whites in Birmingham.

Differences in morbidity patterns by race were evident regardless of asthma history (Tables 30-34). Compared to white children within each community, for any asthma category, black children had significantly higher rates of pneumonia and significantly lower rates for all four other conditions. These differences were consistent for sex and parental education. Among children with a history of asthma, occasional decreases of morbidity rates with age were observed, but no consistent patterns with regard to sex or parental education were evident (these data are not presented).

Morbidity rates among Birmingham black children with "inactive" asthma were generally higher than those in Charlotte whereas the differences were not that consistent for children with "active" asthma (Table 35). For blacks, the increase of one or more episodes of croup among Birmingham "inactive" asthmatic children approached significance ($0.10>p>0.05$) as did the increase of two or more episodes of bronchitis among the similar group in Charlotte (Table 35). Among white children, morbidity rates for "active" asthmatics tended to be higher in Birmingham whereas those among "inactive" asthmatics were higher in Birmingham for only 4 of 10 possible morbidity by number of episode categories (Table 36). Croup rates were significantly higher in Birmingham among "active" asthmatics (Table 37). No other community differences approached significance among white children with a history of asthma. In summary, descriptive city differences in morbidity rates favored the hypothesis among "inactive" black asthmatics and "active" white asthmatics. Statistically significant differences were infrequent, with croup being higher in Birmingham among children of both races and bronchitis in Charlotte approaching significance among black, inactive asthmatic children.

Morbidity rates were significantly increased among residentially stable children of both races in Birmingham, the high particulate exposure community, confirming the study hypothesis. Reported excesses were significant for one or more episodes of "any lower respiratory disease," croup, pneumonia, and hospitalization among black children. Among whites, significant increases in Birmingham were reported for all five morbidity conditions. Model-adjusted morbidity rates were almost always higher in Birmingham than Charlotte. In no case were morbidity rates significantly higher in Charlotte. With regard to age, sex, and education of the head of the household, when significant differences were found, morbidity rates decreased with age, were higher in males and in children from better educated families.

Morbidity reporting was generally comparable to other reported studies for white children. Children with a history of asthma diagnosed by a doctor had two to three times the morbidity rates of their counterparts without such a history.[12,13] Morbidity rates decreased markedly with age, and significant sex differences were due to male excesses.[40,41] Bronchitis and croup were reported much more frequently than pneumonia and hospitalization in white children. Our results differ from British studies in that we found bronchitis to be more frequent among children from families whose parents had at least a high school education, whereas in England bronchitis prevalence was more frequent among lower social class children.[6,7,8,42,43] There are several differences in our study methodology which could explain this seeming disparity. We did not clinically examine our population, limited the recall period to four years, restricted reported illnesses to those diagnosed by a doctor, and used parental education rather than parental occupation as an index of socioeconomic status. Thus, in each community our questionnaire estimated lower respiratory disease morbidity in terms of use of medical care rather than in terms of symptoms, physical findings, or other indices which might be expected to identify illness not coming to the attention of medical care services. Nevertheless, U.S. National Health Survey data show that although chronic bronchitis prevalence varies inversely with the education of the head of the family for persons aged 17 years and over, chronic bronchitis prevalence actually increases with parental education for children under seventeen years of age.[44]

Patterns of lower respiratory disease morbidity reporting among blacks and whites within both communities were similar with regard to age, sex, parental education, and a history of asthma. Such internal consistency suggests that the increased frequency of pneumonia and decreased frequency of bronchitis, croup, and any lower respiratory disease in blacks when compared to whites, was not merely a statistical artifact. Pulmonary function ($FEV_{0.75}$) of black children was significantly lower than white children in a recent survey of Cincinnati elementary schools and for adult males, blacks have a lower FEV_1 and FVC than whites.[44,45] U.S. adult blacks appear to be less susceptible to the effects of cigarette smoke than whites on the basis of mortality, morbidity or decline in pulmonary function with age.[46] However, many other factors besides a true inherent black-white difference were likely to have caused the observed black-white pattern of morbidity found in this study.

For both communities, when compared to white families, black families in this study were less educated, had a lower proportion of two parent families, had a higher proportion of mothers or female guardians aged 50 or above, had larger families, lived in more crowded living conditions, and enjoyed less air conditioning. Differences in medical care utilization would in part explain the differences in morbidity reporting between races. For example, although the number of physician visits per family increased with family size, nonwhites reported fewer visits for each size category than did whites in a recent U.S. survey.[47] Thus, black parents may have utilized a doctor only for more serious illnesses which could explain the higher rate of pneumonia than bronchitis and croup among blacks. Differences in recall, access to care, parental perception of illness, as well as cultural or genetic differences all may be associated with the observed black-white disparity in reported morbidity. Although these questions are of clinical and public health interest, this study was not designed to answer them.

Several other known derminants of childhood respiratory disease morbidity besides ambient air pollution exposures seemed reasonably comparable between the two communities for each race. Family size and composition and household size were similar in both communities. Exposure to sidestream cigarette smoke has been associated with increased acute respiratory morbidity in children.[48] Parental cigarette smoking was slightly more prevalent in Charlotte than in Birmingham which would only minimize any true difference associated with air pollution. Children's personal smoking habits were not ascertained, but there is no a priori reason to believe they would differ markedly in the two cities. Likewise, both communities suffered the impact of the 1968-1969 influenza epidemic. Age, sex, and education of the head of the household, of course, were accounted for in the analysis.

Two factors which certainly could have affected reported morbidity rates in this study are parental recall and the reliability of the questionnaire. Historical morbidity information was restricted to four years for each child rather than his entire life which minimized memory loss regarding older children. Children less than 4 years old were, of course, not at risk for the full four year period, but the age distribution of children aged 1 to 4 years was the same for all city-race groups. In a follow-up study of respiratory illnesses in Sheffield, England, only 52% (81/157) of children with a history of bronchitis or pneumonia at age 5 were reported to have had either illness four years later (illnesses were not restricted to "diagnosed by a doctor").[49] Whereas the effect of decreased parental recall would tend to underestimate the true prevalence of childhood respiratory disease, it would not alter the pattern of our results except in the unlikely circumstances of intercommunity differences in recall.

Morbidity data obtained from household surveys are known to vary with regard to whether the mother or father is interviewed, but in this study, the mother or female guardian was requested to complete the questionnaire whenever possible.[50,51] Our results with regard to respiratory morbidity and age, sex, education of the head of the household, and history of childhood asthma agree quite well with the findings in two previous U.S. surveys using the same questionnaire.[12,13] British studies showing decreased ventilatory function in children with a past history of bronchitis or pneumonia give some confidence in the validity of the questionnaire in assessing the frequency of lower respiratory tract disease.[42,43,49] Further discussion on the validity of the questionnaire is appended (Appendix C).

In this study, excess acute lower respiratory disease morbidity has been associated with exposure to estimated average annual total suspended particulate concentrations of about 133 to 169 $\mu g/m^3$ compared to about 74 to 112 $\mu g/m^3$ among children aged 1 to 12 years. These results do not suggest that the present U.S. Federal primary standard for particulate matter (75 $\mu g/m^3$, annual average, geometric mean) is too stringent because total suspended particulate concentrations in Charlotte were near this standard for several years. Nevertheless, the data cannot tell us if morbidity rates in Charlotte would have been higher compared to a community with even lower total suspended particulate exposure. However, the results clearly associate exposure to elevated particulate matter, in the presence of low sulfur dioxide concentrations, with excess acute lower respiratory morbidity in children.

Children with a history of asthma diagnosed by a doctor were found to have significantly higher rates of "any lower respiratory disease," croup, bronchitis, pneumonia, and hospitalization than children without such a history; morbidity rates were highest among children with a history of asthma active during the past two years and intermediate among children with a history of asthma ever diagnosed by a doctor, but not active during the past two years. Relationships of lower respiratory disease morbidity to age, sex, parental education, race, and ambient air pollution among nonasthmatic children have been reported elsewhere.[22] Morbidity rates of children with asthma showed exactly the same pattern with regard to race, similar, but less striking decreases with age, and no consistent trends in relation to sex or parental education. Lower respiratory disease morbidity for all asthmatic children was comparable to those in two other studies.[12,13] With regard to air pollution, the findings were not as clear as those among nonasthmatic children. Among children with a history of asthma, morbidity rates were higher in Birmingham in half the comparisons studied. Rates for croup were significantly increased in Birmingham among black and white children, although those for bronchitis ($0.10>p>0.05$) were higher in Charlotte among black children only.

Missing information on asthma history was rare (<1% of whites and 1-4% of blacks). In this study, asthma prevalence in children was comparable with published data from Australia, England, The Netherlands, Switzerland, and the United States.[34,49,52-59] The higher proportion of male children with both diagnosed and active asthma was also in accord with expectation. Asthma prevalence among persons aged 17 to 44 years in the U.S. National Health Survey was higher in females and did not vary by race or education of head of family.[58] Our data for asthma among parents were quite similar in nature. The clustering of asthma in families was also expected and would probably have been even stronger if we had asked about other allergic conditions as well as surveying near relatives.[15,60-64] Thus, no major bias regarding the reporting of asthma seems to have been present in the study although no attempts were made to verify positive asthma histories by clinical examination or medical record reviews.

Other factors besides a true increased frequency of acute lower respiratory diseases could have tended to magnify the relative increase we found for children with a history of asthma. Children with a history of asthma would have been more likely to be seen by a physician for an episode of respiratory illness and this possibility would seem most likely for the children with active asthma at the time of the study. When examining an asthmatic child with an acute respiratory illness, a physician probably would be more vigorous in seeking evidence of lower respiratory tract involvement and would be more likely to assume lower respiratory involvement if there were any doubt in his mind. Parental recall would probably be better for children with asthma. Recent evidence suggests a higher frequency of viral infection associated with wheezing in asthmatic children than previously thought.[65,66] Conversely, children who wheeze in association with viral infection appear to have inherited or acquired bronchial hyperactivity.[67] Since there is still question as to whether asthma and wheezy bronchitis in childhood are different diseases, this would increase the association of lower respiratory disease and asthma in children as ascertained in this study.[34,61,62,68-70] Hence, the relative increases of lower respiratory disease in asthmatic children compared to nonasthmatic children from this study, if anything, might be overestimates of the true relative risk.

Croup alone, was significantly increased among asthmatic children in Birmingham. Unlike bronchitis or bronchiolitis with wheezing, croup is not likely to be confused with an attack of asthma by the clinician.[26,71] It is well known that para-influenza viruses Types 1 and 3 are associated with croup with Type 1 being more frequent.[27,72] Infections with parainfluenza Type 3 viruses produce a more variable clinical picture with bronchiolitis or pneumonia predominating in infants, croup in children two to three years of age, and tracheobronchitis above this age. Biannual epidemics of parainfluenza Type 1 virus have been found and at least one longitudinal study has noticed the pattern of parainfluenza Type 3 infections change toward discrete waves from an initial endemic nature.[29] It is possible that an epidemic of parainfluenza Type 1 virus occurring in Birmingham, but not in Charlotte, could have caused the observed increase. Against this is that epidemics of parainfluenza Type 1 virus have occurred simultaneously in places as far apart as North Carolina and Washington whereas other studies have not demonstrated distinct seasonal patterns for this virus.[29] Community differences in diagnostic custom also seems an unlikely explanation.

Measurements of ambient sulfur dioxide and suspended particulates obviously are an index of complex and varied pollution sources.[73] In the two studies of western smelter communities where excess croup was found in exposed asthmatic children, they were more likely to be exposed to frequent fumigations, acid aerosols, and airborne trace metals. These exposure differences might have accounted for the increase of bronchitis and pneumonia observed among exposed asthmatic children in those studies, but not in this one. Although the statistically significant increase in croup could simply be a chance finding, one cannot totally exclude a causal association with particulate air pollution in light of the previous studies.

Against this are the generally inconsistent city differences in morbidity rates along with the significantly $(0.10>p>0.05)$ increased rates of bronchitis in Charlotte black children. One could explain the differences in bronchitis morbidity as a chance deviation with a probability of between 0.05 and 0.10 in a population sample of this size. If they are not due to chance, they may reflect some "protective" effect of particulate air pollution exposure, an explanation which seems even more unlikely. Another possible explanation for the inconsistent city differences in morbidity rates among asthmatic children is that other factors than air pollution such as temperature, dusts, pollens, and other allergens which are known to be related to asthmatic attacks were not measured in this study.[74] Although these factors have been associated with increased asthmatic attacks, and not increased lower respiratory morbidity, in asthmatic children, they, too, may increase the risk of lower respiratory disease in asthmatic children. Alternately, some of the reported acute lower respiratory disease in these asthmatic children may, in fact have been episodes of asthma. Further studies will be necessary to fully clarify the relationship of air pollution exposure and morbidity reporting among children with a history of asthma.

TABLE 15. ESTIMATED[a] POLLUTANT EXPOSURE LEVELS IN CHARLOTTE, NORTH CAROLINA (INTERMEDIATE EXPOSURE) AND BIRMINGHAM, ALABAMA (HIGH EXPOSURE): 1960-1971

Pollutant	Community	Estimated Pollutant Concentrations, µg/m³ 1960-63 Average	1964-67 Average	1968-71 Average	1960-71 Average	National Air Quality Std. Annual Average
Total Suspended Particulates	Charlotte	107	93	79	93(74-112)[b]	75 µg/m³ (geometric mean)
	Birmingham	168	159	139	155(133-169)	
Sulfur Dioxide	Charlotte	17	16	17	17(13-20)	80 µg/m³ (0.03 ppm)
	Birmingham	<25	11	13	15(6-<25)	
Suspended Sulfates	Charlotte	6	9	10	8(4-10)	None
	Birmingham	10	11	14	10(9-16)	
Suspended Nitrates	Charlotte	2	2	1	2(1-3)	None
	Birmingham	2	3	3	2(2-3)	

[a] All values obtained from reference 14 and based on both measured and estimated values. Twenty-four hour integrated estimates of concentrations were measured expressed in micrograms per cubic meter. For each year, the average of all daily estimates was computed. For periods of several years the average of the individual years is tabulated. For the period 1960 to 1971 the average for the 12-year period is shown together with the range of the 12 individual years.

[b] Range in parentheses

TABLE 16. TOTAL NUMBER OF QUESTIONNAIRES DISTRIBUTED AND RESPONSE RATE AMONG STUDY FAMILIES*

Community	Race	Questionnaires Distributed	
		Returned, %	Total Number
Charlotte	Total	88	3448
	Black	84	1083
	White	89	2365
Birmingham	Total	95	2941
	Black	95	1382
	White	94	1559

*Refers to number of families (households) of children aged one to twelve years (of whom only children aged six to twelve received questionnaires at school).

TABLE 17a. CHILDREN AGED ONE TO TWELVE EXCLUDED FROM ANALYSIS BECAUSE OF MISSING INFORMATION, BY CITY AND RACE*

Community	Race	Total Children Excluded		Percent Excluded by Specific Category			
		Number	Percent	Morbidity Only	Asthma** History Only	Age, Sex, Residence Duration or Education of Head of Household Only	Two or More Missing Categories
Charlotte	Black	294/2208	13.3	6.7	4.2	1.7	0.7
	White	175/3964	4.4	2.8	0.8	0.3	0.5
Birmingham	Black	278/3010	9.2	7.2	1.0	1.0	0.0
	White	113/2611	4.3	3.2	0.3	0.8	0.0

*Refers to number of children aged one to twelve (of whom only children aged six to twelve received questionnaires at school).

**Missing information about history of asthma was obtained from respondents by telephone only in Birmingham.

TABLE 17b. CHILDREN AGED 1 TO 12 YEARS EXCLUDED FROM ANALYSIS BECAUSE OF MISSING* INFORMATION

Community	Race	Percent Excluded By Asthma History	
		Asthma, (ever diagnosed)	Not Asthma (never diagnosed)
Charlotte	Black	11.1	12.9
	White	5.4	4.3
Birmingham	Black	13.2	9.0
	White	4.7	4.3

*Missing information on morbidity, age, sex, education of the head of household, or duration of residence. About 4% of black children in Charlotte and <1% of all other children were excluded because of missing information on history of asthma.

TABLE 18a. DURATION OF RESIDENCE, EDUCATION OF FATHERS, PRESENCE OF PARENTS OR GUARDIANS AND PARENTAL SMOKING HABITS AMONG STUDY FAMILIES, BY CITY AND SEX

Community	Race	Three or More Years Residence in Community, %	Fathers Completing High School, %		Presence of Parents or Guardians, %		Current Cigarette Smokers, %	
			< 39 years	> 40 years	Both	Mother Only	Mothers	Fathers
Charlotte	Black(744)[a]	92	55	26	63	33	43	63
	White(1731)	89	65	57	85	13	48	64
Birmingham	Black(1094)	95	58	33	71	27	34	56
	White(1144)	87	65	49	83	15	43	66

[a] Number of families with three or more years residence in community

TABLE 18b. DURATION OF RESIDENCE, EDUCATION OF FATHERS, PRESENCE OF PARENTS OR GUARDIANS AND PARENTAL SMOKING HABITS AMONG STUDY FAMILIES WITH ONE OR MORE CHILDREN WITH A HISTORY OF ASTHMA

Community	Race	Three or More Years Residence in Community	Fathers Completing High School		Presence of Parents or Guardians		Current Cigarette Smokers	
			<39 years	>40 years	Both	Mother Only	Mothers	Fathers
Charlotte	Black(89)^{a}	87%	70%	41%	63%	32%	46%	56%
	White(209)	89%	71%	48%	85%	13%	51%	65%
Birmingham	Black(100)	92%	44%	27%	77%	22%	38%	45%
	White(184)	87%	65%	45%	85%	13%	48%	71%

[a] Number of families with three or more years residence in community.

TABLE 19a. MATERNAL AGE AND HOUSEHOLD CHARACTERISTICS OF STUDY FAMILIES, BY CITY AND RACE

		Age of Mother or Female Guardian		Persons per Household[a]		Number of Rooms per Household		Air Conditioning in Households	
Community	Race	Median	>50 years	Median	>5	Median	>5	Window Only	Central
Charlotte	Black	34 yrs	10%	4	35%	5	69%	28%	6%
	White	34 yrs	4%	3	9%	5	83%	56%	7%
Birmingham	Black	37 yrs	13%	4	39%	6	73%	37%	6%
	White	34 yrs	4%	3	10%	6	81%	60%	8%

[a] Excluding parents or guardians

TABLE 19b. MATERNAL AGE AND HOUSEHOLD CHARACTERISTICS OF STUDY FAMILY WITH ONE OR MORE CHILDREN WITH A HISTORY OF ASTHMA

		Age of Mother or Female Guardian		Persons per Household		Number of Rooms per Household		Air Conditioning in Households	
Community	Race	Median	>50 years	Median	>5	Median	>5	Window Only	Central
Charlotte	Black	33 yrs	15%	4	45%	5	76%	26%	14%
	White	35 yrs	4%	3	15%	5	85%	60%	4%
Birmingham	Black	37 yrs	9%	4	46%	6	78%	35%	8%
	White	34 yrs	2%	3	14%	6	85%	62%	6%

[a] Excluding parents or guardians

TABLE 20. CHILDREN WITHOUT A HISTORY OF ASTHMA, BY CITY, RACE, SEX AND AGE*

		Number of Children			
Community	Race and Sex	1-4 Years	5-8 Years	9-12 Years	1-12 Years
Charlotte	Black Female	134	331	398	863
	Male	120	296	378	794
	White Female	206	583	753	1542
	Male	243	599	777	1619
Birmingham	Black Female	206	480	553	1239
	Male	205	503	549	1257
	White Female	143	396	454	993
	Male	148	388	456	992
All Children		1405 (15%)	3576 (38%)	4318 (47%)	9299 (100%)

*Excludes children with a history of asthma, children with less than three years residence duration and children with missing information on morbidity, history of asthma, age, race, sex, education of head of household or duration of residence.

TABLE 21. CHILDREN WITH A HISTORY OF ASTHMA BY CITY, RACE, SEX AND AGE*

		Number of Children			
Community	Race and Sex	1-4 Years	5-8 Years	9-12 Years	1-12 Years
Charlotte	Black Female	0	17	16	33
	Male	2	16	25	43
	White Female	7	33	28	68
	Male	15	36	53	104
Birmingham	Black Female	1	17	16	34
	Male	3	21	22	46
	White Female	9	14	30	53
	Male	24	38	51	113
All Children		61 (12%)	192 (39%)	241 (49%)	494 (100%)

*Excludes children without a history of asthma, children with less than three years residence duration, and children with missing information on morbidity, history of asthma, age, race, sex, education of head of household or duration of residence.

TABLE 22. HISTORY OF ASTHMA EVER DIAGNOSED BY A DOCTOR: PREVALENCE IN CHILDREN BY AGE, SEX, RACE AND COMMUNITY

Age in Years	Asthma Ever Diagnosed, %							
	Charlotte				Birmingham			
	Black		White		Black		White	
	Female	Male	Female	Male	Female	Male	Female	Male
1-4	0.0	1.6	3.3	5.9	0.5	1.5	5.9	14.0
5-8	4.9	5.6	5.3	5.7	3.4	4.0	3.4	9.1
9-12	3.9	6.2	3.7	6.6	2.8	4.0	6.2	10.0
All Ages	3.7	5.2	4.3	6.1	2.7	3.6	5.1	10.3

TABLE 23. HISTORY OF ASTHMA ACTIVE DURING THE PAST TWO YEARS: PREVALENCE IN CHILDREN BY AGE, SEX, RACE, AND COMMUNITY

Age in Years	Asthma Active in Past Two Years, %							
	Charlotte				Birmingham			
	Black		White		Black		White	
	Female	Male	Female	Male	Female	Male	Female	Male
1-4	0.0	0.8	2.8	4.3	0.0	0.5	3.9	9.9
5-8	2.3	4.8	2.1	2.7	2.2	2.3	1.2	4.7
9-12	1.2	3.5	1.7	3.7	1.6	2.1	1.7	4.3
All Ages	1.5	3.6	2.0	3.4	1.6	1.9	1.8	5.3

TABLE 24. PERCENT OF CHILDREN WITH ACTIVE ASTHMA AMONG CHILDREN WITH ASTHMA EVER DIAGNOSED BY A DOCTOR, BY AGE, SEX, RACE, AND COMMUNITY

Age in Years	Percent of Active to all Asthmatic Children							
	Charlotte				Birmingham			
	Black		White		Black		White	
	Female	Male	Female	Male	Female	Male	Female	Male
1-4	-*	50	86	73	0	33	67	71
5-8	47	98	39	47	65	57	36	51
9-12	31	56	33	56	56	52	27	43
All Ages	39	70	46	56	59	53	36	52

*No children in this cell, therefore rate is indeterminate.

TABLE 25. HISTORY OF ASTHMA DIAGNOSED BY A DOCTOR: PREVALENCE IN FAMILIES* OF ELEMENTARY SCHOOL CHILDREN BY RACE, SEX AND COMMUNITY

				History of Asthma		
Community Exposure	Race	Sex	Sample Size	Yes, Ever Diagnosed	Yes, Active Past 2 Years	Active / Ever Diagnosed
Charlotte	Black	Mother	342	7.6%	4.7%	61.5%
		Father	342	4.7%	1.5%	31.3%
	White	Mother	1004	8.5%	4.4%	51.8%
		Father	1002	9.1%	3.5%	38.5%
Birmingham	Black	Mother	792	3.9%	2.3%	58.1%
		Father	788	2.0%	0.5%	25.0%
	White	Mother	1053	5.6%	3.5%	62.7%
		Father	1046	4.9%	2.0%	41.2%

*Only families with both parents present included.

TABLE 26. ASTHMA EVER DIAGNOSED BY A DOCTOR: PREVALENCE IN CHILDREN IN RELATION TO PREVALENCE IN THEIR PARENTS*

Asthma Ever Diagnosed in Parents	One or More Children With Asthma Ever Diagnosed, %				
	Charlotte		Birmingham		All Families, % (Total Families)
	Black	White	Black	White	
Neither	10.9	10.2	8.7	14.5	11.1 (3398)
One	38.2	28.9	35.7	32.6	32.0 (300)
Both	66.7	77.8	100.0	83.0	80.0 (20)

*Only families with both parents present included.

Table 27. ASTHMA EVER DIAGNOSED BY A DOCTOR: PREVALENCE IN CHILDREN IN RELATION TO ASTHMA ACTIVITY IN THEIR PARENTS*

Asthma Activity** of Parents	One or More Children With Asthma Diagnosed, %			
	Charlotte		Birmingham	
	Black	White	Black	White
Never diagnosed	10.9	10.2	8.7	14.5
Diagnosed, not active	13.3	24.7	39.1	31.0
Active, past two years	57.9	33.9	31.6	34.0

*Only families with both parents present, but not with both parents having asthma included.

**Never diagnosed - never diagnosed in either parent
Diagnosed, not active - diagnosed in one or both parents, not active in either
Active, past two years - active in one or both parents in the past two years

TABLE 28. FOUR YEAR FREQUENCY OF EACH MORBIDITY CONDITION BY NUMBER OF EPISODES AND COMMUNITY: MODEL ADJUSTED RATES FOR BLACK CHILDREN AGED 1 TO 12 YEARS

Morbidity Condition	Number of Episodes	Model Adjusted Rates*		$\frac{\text{Birmingham}}{\text{Charlotte}}$
		Charlotte	Birmingham	
"Any Lower Respiratory Disease"	≥1	18.7%	20.0%	1.07
	≥2	10.1%	10.4%	1.03
Croup	≥1	7.0%	7.1%	1.01
	≥2	2.4%	2.5%	1.04
Bronchitis	≥1	9.2%	8.4%	0.91
	≥2	3.6%	3.0%	0.83
Pneumonia	≥1	9.5%	12.8%	1.35
	≥2	4.1%	4.9%	1.20
Hospitalization	≥1	2.6%	3.6%	1.38
	≥2	Rate too low to fit model.		--

*Age-sex-education head of household adjusted rates for non-asthmatic children with three or more years residence duration from saturated linear model for categorical data (cf. Table 11).

TABLE 29. FOUR YEAR FREQUENCY OF EACH MORBIDITY CONDITION BY NUMBER OF EPISODES AND COMMUNITY: MODEL ADJUSTED RATES FOR WHITE CHILDREN AGED 1 TO 12 YEARS

Morbidity Condition	Number of Episodes	Model Adjusted Rates*		Birmingham/Charlotte
		Charlotte	Birmingham	
"Any Lower Respiratory Disease"	≥1	28.7%	33.6%	1.17
	≥2	16.2%	20.3%	1.25
Croup	≥1	13.5%	14.5%	1.07
	≥2	5.6%	7.4%	1.32
Bronchitis	≥1	19.3%	24.2%	1.25
	≥2	8.3%	13.6%	1.64
Pneumonia	≥1	6.8%	8.5%	1.25
	≥2	Rate too low to fit model		--
Hospitalization	≥1	3.0%	4.6%	1.53
	≥2	Rate too low to fit model.		--

*Age-sex-education head of household adjusted rates for non-asthmatic children with three or more years residence duration from saturated linear model for categorical data (cf. Table 4).

TABLE 30. "ANY LOWER RESPIRATORY DISEASE": FOUR YEAR FREQUENCY BY HISTORY OF ASTHMA DIAGNOSED BY A DOCTOR

Race	History of Asthma	"Any Lower Respiratory Disease*", %			
		One or More Episodes		Two or More Episodes	
		Charlotte	Birmingham	Charlotte	Birmingham
Black	Never diagnosed	16.2	18.8	8.6	9.7
	Diagnosed, not active	42.4	48.6	24.2	31.4
	Active, past two years	62.8	60.0	44.2	44.4
White	Never diagnosed	27.1	31.8	15.4	19.9
	Diagnosed, not active	55.6	47.7	43.2	33.0
	Active, past two years	83.5	80.8	70.3	71.8

*Crude rates of children aged 1 to 12 years.

TABLE 31. CROUP: FOUR YEAR FREQUENCY BY HISTORY OF ASTHMA DIAGNOSED BY A DOCTOR

Race	History of Asthma	Croup*, %			
		One or More Episodes		Two or More Episodes	
		Charlotte	Birmingham	Charlotte	Birmingham
Black	Never diagnosed	6.5	6.9	1.8	2.5
	Diagnosed, not active	15.1	37.2	3.0	8.9
	Active, past two years	23.3	21.9	14.0	8.6
White	Never diagnosed	12.4	14.4	5.3	7.5
	Diagnosed, not active	28.4	32.9	11.1	18.1
	Active, past two years	23.1	51.4	19.8	42.4

*Crude rates of children aged 1 to 12 years.

TABLE 32. FOUR YEAR FREQUENCY BY HISTORY OF ASTHMA DIAGNOSED BY A DOCTOR

Race	History of Asthma	Bronchitis*,%			
		One or More Episodes		Two or More Episodes	
		Charlotte	Birmingham	Charlotte	Birmingham
Black	Never diagnosed	7.9	7.8	2.9	2.8
	Diagnosed, not active	24.2	14.3	15.1	2.9
	Active, past two years	53.5	48.8	32.6	24.4
White	Never diagnosed	18.4	23.5	8.5	8.6
	Diagnosed, not active	48.1	37.5	30.8	22.7
	Active, past two years	76.9	78.2	60.4	64.1

*Crude rates of children aged 1 to 12 years.

TABLE 33. PNEUMONIA: FOUR YEAR FREQUENCY BY HISTORY OF ASTHMA DIAGNOSED BY A DOCTOR

Race	History of Asthma	Pneumonia*, %			
		One or More Episodes		Two or More Episodes	
		Charlotte	Birmingham	Charlotte	Birmingham
Black	Never diagnosed	8.4	12.2	3.7	4.8
	Diagnosed, not active	15.1	25.7	6.0	8.6
	Active, past two years	32.6	31.2	7.0	15.6
White	Never diagnosed	6.3	7.7	1.9	2.4
	Diagnosed, not active	12.3	6.9	2.4	4.6
	Active, past two years	23.1	24.3	9.9	8.9

*Crude rates of children aged 1 to 12 years.

TABLE 34. HOSPITALIZATION: FOUR YEAR FREQUENCY BY HISTORY OF ASTHMA DIAGNOSED BY A DOCTOR

Race	History of Asthma	Hospitalization*, %			
		One or More Episodes		Two or More Episodes	
		Charlotte	Birmingham	Charlotte	Birmingham
Black	Never diagnosed	1.9	3.2	0.4	0.3
	Diagnosed, not active	6.1	14.3	0.0	5.7
	Active, past two years	2.3	6.7	0.0	4.4
White	Never diagnosed	2.3	4.5	0.3	0.8
	Diagnosed, not active	3.7	3.4	0.0	1.1
	Active, past two years	8.8	16.7	0.0	1.3

*Crude rates of children aged 1 to 12 years.

TABLE 35. FOUR YEAR FREQUENCY OF MORBIDITY AMONG BLACK ASTHMATIC CHILDREN BY HISTORY OF ASTHMATIC ACTIVITY

Morbidity Condition	Number of Episodes	Morbidity Rates*, by Asthma Activity, %			
		"Inactive" Asthma		"Active" Asthma	
		Charlotte	Birmingham	Charlotte	Birmingham
"Any Lower Respiratory Disease"	≥1	42.4	48.6	62.8	60.0
	≥2	24.2	31.4	44.2	44.4
Croup	≥1	15.1	37.2^{d}	23.3	21.9
	≥2	3.0	8.9	14.0	8.6
Bronchitis	≥1	24.2	14.3	53.5	48.8
	≥2	15.1	2.9^{d}	32.6	24.4
Pneumonia	≥1	15.1	25.7	32.6	31.2
	≥2	8.0	8.6	7.0	15.6
Hospitalization	≥1	6.1	14.3	2.3	6.7
	≥2	0.0	5.7	0.0	4.4

*Crude rates of children aged 1 to 12 years

a - $p<0.001$; b - $p<0.01$; c - $p<0.05$; d - $0.10>p>0.05$

Probabilities for a two-tailed test of significance, viz. the probability for morbidity excesses in either city as large or larger than those observed given that there is no difference.

TABLE 36. FOUR YEAR FREQUENCY OF MORBIDITY AMONG WHITE ASTHMATIC CHILDREN BY HISTORY OF ASTHMATIC ACTIVITY

Morbidity Condition	Number of Episodes	Morbidity Rates*, by Asthma Activity, %			
		"Inactive" Asthma		"Active" Asthma	
		Charlotte	Birmingham	Charlotte	Birmingham
"Any Lower Respiratory Disease"	≥1	55.6	47.7	83.5	80.8
	≥2	43.2	33.0	70.3	71.8
Croup	≥1	28.4	32.9	23.1	51.4[a]
	≥2	11.1	18.1	19.8	42.4[b]
Bronchitis	≥1	48.1	37.5	76.9	78.2
	≥2	30.8	22.7	60.0	64.1
Pneumonia	≥1	12.3	6.9	23.1	24.3
	≥2	2.4	4.6	9.9	8.9
Hospitalization	≥1	3.7	3.4	8.8	16.7
	≥2	0.0	1.1	0.0	1.3

*Crude rates of children aged 1 to 12 years.

a - $p \leq 0.001$; b - $p \leq 0.01$; c - $p \leq 0.05$; d - $0.10 \geq p \geq 0.05$

Probabilities for a two-tailed test of significance, viz. the probability for morbidity excesses in either city as large or larger than those observed given that there is no difference (cf. Table 37).

TABLE 37. CHI-SQUARE AND SIGNIFICANCE LEVELS: COMMUNITY DIFFERENCES IN LOWER RESPIRATORY DISEASE AMONG CHILDREN WITH A HISTORY OF ASTHMA AND THREE OR MORE YEARS RESIDENCE DURATION

Race	Asthma Category	Any LRD		Croup		Bronchitis		Pneumonia		Hospitalization	
		≥1	≥2	≥1	≥2	≥1	≥2	≥1	≥2	≥1	≥2
Black	Inactive (33,35)*	0.70	0.15	3.17[d]	0.95	0.54	3.00[d]	0.60	0.40	0.52	1.26
	Active (43,45)	<0.01	0.04	0.01	0.60	0.05	0.36	0.01	0.87	0.97	1.27
White	Inactive(81,88)	0.75	1.47	0.22	1.16	1.54	0.85	0.93	0.54	0.25	<0.01
	Active(91,78)	0.07	<0.01	13.29[a]	9.07[b]	<0.01	0.11	<0.01	<0.01	1.72	<0.01

*Sample sizes for Charlotte and Birmingham in parentheses.

a - $p \leq 0.001$
b - $p \leq 0.01$
c - $p \leq 0.05$ For each term in the model, the probability for a two-tailed test of statistical significance
d - $0.10 > p > 0.05$

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APPENDIX A

REVIEW OF THE LITERATURE

Unlike adults, children are generally not subjected to occupational pollution exposures or to "self-pollution" by smoking tobacco. Children also are less likely to have experienced several different long term air pollution exposures related to residential mobility. Surveys of acute morbidity in children would be least likely to be confounded with symptoms of chronic disease because of the relatively low frequency of chronic disease among them. These advantages, first noted a decade ago[75] make children an excellent group in which to study adverse health effects associated with exposure to ambient air pollutants.

Bronchial asthma, a disorder of the lower respiratory tract, is characterized by periodic attacks of obstructive expiratory dyspnea resulting in wheezing, cough, and dyspnea. There is not one pathognomonic clinical or laboratory picture for bronchial asthma, but the American Thoracic Society has proposed the following definition: "A disease characterized by an increased responsiveness of the trachea and bronchi to various stimuli and manifested by widespread narrowing of the airways which changes in severity either spontaneously or as a result of therapy." The definition goes on to point out that the term "asthma," as so defined, may occur in subjects with other bronchopulmonary or cardiovascular diseases, but in these instances the airway obstruction is not causally related to these diseases.[15] As defined in this sense, asthma is a chronic lower respiratory disease which varies in duration, severity, and frequency of attacks. This review, therefore, is organized as follows: the effects of air pollution on acute lower respiratory disease in nonasthmatic children, the effects of air pollution on acute lower respiratory disease in asthmatic children, and the effects of air pollution on the incidence of asthma and frequency of asthma attacks in afflicted children. The major portion of this review will deal with the first topic, since the bulk of the published literature to date concerns air pollution and nonasthmatic children. Pertinent studies of air pollution and pulmonary function, per se, in children, will be discussed in relation to the above topics.

Air Pollution and Acute Lower Respiratory Disease: Nonasthmatic Children

Toyama studied two groups of about 100 school chilren, aged 10 and 11 in Kawasaki, Japan.[9] Children in the more polluted area had a higher frequency of occasional nonproductive cough, of a sense of mucous membrane irritation, frequent mucous secretion by medical examination, and lower peak expiratory flow rates (PFR). No statistically significant differences in school abscences or total vital capacity were observed. Watanabe studied about 150 fourth grade children in each of three schools (one with low and two with moderate pollution) in Osaka City.[76] He found that PFR decreased more in winter for children in polluted areas, but no difference was found in the prevalence of cold symptoms measured as: "I have a cold, cough, sore throat, runny nose, etc."

Anderson and Larsen studied the health effects of kraft pulping mills upon children in Canada. They studied a total of about 750 first graders in two polluted and one control community.[77] They found no conclusive differences between communities for school absences or the incidence of respiratory illnesses, but did find tonsillectomy, inflamed eyes, headache, feverishness and nausea to be more frequent in the polluted area. Anderson and Kinnis did find the PFR in the polluted community to be significantly lower than expected when compared to the less polluted community.[78] Ferris studied a total of about 700 first and second graders in Berlin, New Hampshire (about 60 to 150 in each of seven schools in two successive school years).[79] He concluded school absences did not differ among schools but that measurements of pulmonary function, PFR and FEV forced vital capacity in one second ($FEV_{1.0}$) did show significant differences which could have been due to air pollution.

Biersteker and van Leeuwen studied about 500 children in a "wealthy" and a "poor" part of Rotterdam.[54] In the wealthy district, the mean smoke concentration in the winter was $50\ \mu g/m^3$ and the mean sulfur dioxide level was $200\ \mu g/m^3$ while these values were about 50% higher in the poor district. They found that PFR, height, and weight were all lower in the poor district and concluded that lower peak flow rates were not due solely to differences in air pollution. Not only were their samples poorly matched by social class, but they included no estimates of social class in their study. Also, one would not expect to see large differences in PFR with the differences in mean pollutant levels that they estimated for only one season.

Shy, et al., studied the $FEV_{0.75}$ of schoolchildren living in neighborhoods with high and low pollution in Cincinnati, Ohio and New York City, New York.[44] Sulfur dioxide, total suspended particulate matter and suspended sulfates were measured daily. Past exposures were estimated for the three New York communities. In the Cincinnati study, performance of children in polluted neighborhoods improved during seasons of low pollution, but not to the level of their counterparts in low pollution neighborhoods. In New York, children aged 9-13 years in the high pollution neighborhood had a poorer $FEV_{0.75}$ performance whereas those aged 5-8 years did not. Since pollution levels had decreased in the two high pollution exposure communities, the authors concluded that the higher past exposures had caused persistent $FEV_{0.75}$ performance decrements in the older children. Chapman, et al., have recently reported a study of $FEV_{0.75}$ in black and white schoolchildren living in Birmingham, Alabama and Charlotte, N.C., the former being a community with a higher particulate exposure than the latter.[23] Both black and white schoolchildren of both sexes living in Birmingham had a poorer average $FEV_{0.75}$ performance than their counterparts in Charlotte with exposure to less pollution. All studies included current air pollution monitoring (of varying types) but only those of Anderson, et al. and Ferris noted "mobility" of children.

Thus alterations in pulmonary function (PFR and $FEV_{1.0}$) did appear to be related to differences in both short term and long term pollution exposure. None of these studies, except Shy's, ascertained the children's asthma history nor did they look at PFR or $FEV_{1.0}$ as a function of past respiratory illness histories in children. Pulmonary function in older children and young adults has been shown to vary inversely with cigarette smoking.[51,80,81] Almost none of the published studies of air pollution and pulmonary function in children have ascertained their smoking habits (due in great part to the difficulty of obtaining an accurate smoking history). Theoretically, the effects of air pollution upon pulmonary function in older children (about 10 or more years of age) could be confounded with those of cigarette smoking if a much higher proportion of children in one area were cigarette smokers than in another area. This possibility seems unlikely, especially since the children in most of the studies were comparable by social class.

Douglas and Waller studied about 4000 children born during the first week of March 1946 through 1961 and whose families did not move (80% of total sample) classifying them into four residence areas of increasing pollution.[6] Pollutant levels were estimated from domestic coal consumption on four areas for the year ending May 1952. Mothers were interviewed about colds, lower respiratory infection (bronchitis, bronchopneumonia, or pneumonia), and recorded hospitalizations when their children were two years old; and colds and recorded hospitalizations when their children were four years old. School doctors examined the children at ages 6, 7, 11 and 15 years. At these times the mothers were asked about the children's colds, coughs, and hospital admissions. Doctors recorded rales, rhonchi, or other abnormal chest sounds and described the upper respiratory passages and the tonsils during these examinations. Health interviewers and physicians were not specially trained for this study. No significant differences among these areas were found for the following: first cold before age of 10 months; more than two colds between 21 and 23 months, frequent or continual colds between 46 and 51 months; mucopurulent discharge at 6 or 7 years of age; ears discharging at or before 2 years, 4 years, 6 years; tonsils removed or needing removal at 6 years, 11 years and 15 years.

Lower respiratory tract illness followed the pollution gradient during the first and second year of life for boys and girls of middle class and manual working class families. Hospital admissions for lower respiratory infections during the first five years of life, but not acute upper respiratory infections or tonsillitis and tonsillectomy, also followed the pollution gradient. The prevalence of rales or rhonchi recorded on one or more and two or more occasions as well as at age 15 also followed the pollution gradient. With regard to school absences, excess (1.5 standard deviations above the average for all schoolchildren) episodes significantly followed the pollution gradient in the highest pollution community. Among the three causes for the longer absences, i.e. more than one week's duration, colds and influenza were equal in the four areas, but absences due to bronchitis increased with the pollution gradient ($0.2>p>0.1$). Douglas and

Waller interpreted the school absence findings as suggesting that the level of air pollution is related to the number of short term absences from school rather than to the total amount of absence. Unfortunately, in the studies of Toyama (1964), Anderson and Larsen (1966), Anderson and Kinnis (1967), and that of Ferris (1970), the data were not analyzed in this manner.[9,77-79] Hence school absences appear to be an indicator of pollution, but must be analyzed with regard to specific causes for absences and number of episodes as well as total time away from school.

Lunn, Knoweldon, and Handyside (1967) studied a total of 819 children with an average age of 5 years, 4 months, in four areas of increasing pollution exposure in Sheffield, England.[7] Sample sizes were 413, 194, 130 and 82 in each area from low to high pollution and comprised the whole first year infant intake of eight local authority schools. Atmospheric pollution (smoke and sulfur dioxide) was monitored from 1964 to 1966 in all study areas. Duration of residence in the community was not ascertained. Parents of school children completed a questionnaire sent out and returned via the school and were asked whether the child suffered from a persistent or frequent cough, more than three colds a year, earache or ear discharge, sore throats or tonsillitis, and whether colds usually went to the child's chest. A history of asthma was not obtained. Two or three weeks later, children were examined for palpable tonsillar lymph glands, mucopurulent nasal discharge, scarring or perforation of the eardrums, tonsillar enlargement and a doctor recorded the $FEV_{0.75}$ and FVC (forced vital capacity). Mucopurulent nasal discharge and a history of three or more colds going to the chest were significantly higher in the polluted communities. A history of persistent or frequent cough significantly followed the pollution gradient. The history of lower respiratory tract disease (pneumonia or bronchitis) also followed the pollution gradient.

The average height adjusted $FEV_{0.75}$ and FVC varied inversely with the pollution gradient. This study also found that the mean $FEV_{0.75}$ and FVC were significantly lowered in children with a history of persistent or frequent cough, a history of colds going to the chest or a history of one of two episodes of lower respiratory tract illness. Children with a history of three or more episodes had the lowest $FEV_{0.75}$ and FVC values. Lunn, et al., concluded that both chronic upper respiratory infections and lower respiratory infections were related to air pollution exposure.[7] They suggested that one reason Douglas and Waller did not show a similar relationship between air pollution and upper respiratory disease was the less precise estimates of air pollution exposure. A much more obvious reason is that the studies looked at different-aged people and measured different things. Lunn, et al., obtained a history of "more than three colds a year" in children aged 5 1/2 years (no time period was given), whereas Douglas and Waller only obtained "more than two colds between 21 and 23 months, frequent or continual colds between 46 and 51 months, or mucopurulent discharge at age 6 and 7 years."[6,7] Thus, the methods of ascertainment are not comparable, and this seems to be the most likely reason for the difference between the two studies with regard to upper respiratory infections. Lunn, et al., stressed that their study was done in the summertime when pollution levels were low. Because of this, they also concluded that a persistent pattern of respiratory disability had appeared at an early age.

Lunn, Knowelden and Roe reported the followup findings on the original group of 5 year olds reexamined four years later.[49] This paper also contained previously unpublished data on a group of 11 year old children who were examined at the same time as the original 5 year old cohort. A history of lower respiratory tract illnesses, pneumonia, and bronchitis at some time in the past was given less frequently by the 11 year old than by the 5 year old children even though the former had twice as long to suffer these illnesses. Lunn, et al. were surprised at these results, but they were most likely due to memory loss. Nevertheless, the 11 year olds also showed an excess of respiratory illness in the more polluted areas, but to a lesser degree than the 5 year olds. The children at 9 years of age also gave a less frequent history of three or more colds, persistent cough or colds going to the chest than at 5 years. Atmospheric pollution levels had also fallen during this time period. When the original 5 year olds were seen four years later, no significant differences between pollution exposure and respiratory morbidity were found at age 9 suggesting that the absence of differences in pollution exposure was associated with this.

Holland, et al. studied almost 11,000 children aged 5-14 plus years in four areas of increased air pollution exposure.[8] No quantitative estimates of air pollution were given. Parents of the children completed a questionnaire on the respiratory disease history of their children and were examined by one of the trained medical officers. They found that area of residence, social class, family size and a past history of pneumonia, bronchitis, or asthma were related to the childrens' PFR in an independent and additive fashion. Colley and Reid studied over 10,000 children aged 6-10 years in contrasting urban and rural areas in England and Wales.[43] Winter mean sulfur dioxide levels were given, but monitoring sites were present in only two out of five rural areas. Parents completed a questionnaire on respiratory symptoms and illnesses for their children at home, and the children were subsequently examined by one of 30 school medical officers following a uniform protocol. Chronic cough and a past history of bronchitis increased from less polluted rural to more polluted urban areas only in children of families of social classes IV and V. Upper respiratory tract infections measured as nasal obstruction or ear discharge, perforation or scarring did not follow the pollutant gradient although the rates for more serious ear disease were highest in the two high pollution cities. Little difference was found between the PFR of the five areas. Unfortunately, the poor documentation of air pollution exposure weakens the utility of the otherwise excellent papers of Holland, et al. and Colley and Reid.[8,43]

Manzhenko studied about 3000 children who had resided in two school districts in Irkutsk, Russia with high and low pollution levels for 5 years or more (750 children lived in the low community).[10] Sulfur dioxide, dust, and tarry substances were measured during the year 1960-61. The incidence of upper respiratory tract infections in both communities was determined by reviewing the records of school medical examinations (carried out by the district pediatrician). He found upper respiratory tract infections including chronic tonsillitis, chronic rhinitis, chronic sinusitis and upper respiratory tract catarrh all to be significantly increased in the higher pollution district. He also found more abnormal X-rays (13% versus 2% for 948 and 250 children, respectively) manifested as hilar changes only, hilar changes plus findings in the lungs, and marked hilar and pulmonary findings. No information on past medical history, social class, or technique of examination was given. However, monthly mean sulfur dioxide concentrations were quite high, ranging from 0.11 to 1.99 mg/m^3 during the year of the study. Sulfur dioxide was done by the "aspiration" method, so it is not clear if it was a gaseous determination or estimated from sulfation fallout. Also, the nature of the "tarry substances" was not described.

Recently, four studies of acute respiratory disease in U.S. children have been published, initially in a summary article and subsequently in four separate papers in a monograph.[11,12,13,82,83] Two retrospective surveys of acute lower respiratory disease were done in several smelting communities in the Salt Lake Basin and the Rocky Mountains by Nelson, et al. and Finklea, et al.[12,13] Current exposure to sulfur dioxide, total suspended particulate matter and suspended sulfates was monitored and past exposures were estimated from smelter production or emissions and previous aerometric data. In Utah, four communities representing one "Low", two "Intermediate" and one "High" air pollution exposure were studied. In the Rocky Mountain study, five communities were studied and pooled into "High" exposure (two communities) and "Low" exposure (three communities) for statistical hypothesis testing. Parents completed the questionnaire and returned it via the schools answering questions about a history of pneumonia, croup, bronchitis, bronchiolitis or other deep chest infections in children aged 1 to 12 years during the three years prior to the study. Duration of residence in the community, parental smoking habits, occupational exposure and education of the head of household were also obtained. Analyses were restricted to children without a history of asthma from families with three or more years residence duration (7763 children in Utah and 4305 children in the Rocky Mountain study). Significant differences with respect to pollution were found in both studies. In the Utah study, one or more and two or more episodes of "any lower respiratory disease" (any LRD, a combined category), croup, and bronchitis were significantly increased. In the Rocky Mountain study, two or more episodes of "any LRD" and croup were significantly increased in the high pollution communities. Differences for one or more episodes of "any LRD" and two or more episodes of bronchitis approached statistical significance ($0.10>p>0.05$). Rates for pneumonia and hospitalization did not differ among the communities. Respiratory morbidity decreased with age and tended to be increased in males. Any LRD, croup and bronchitis tended to be more frequent in children from households with a high school or better education; the converse was true for pneumonia and hospitalization.

Finklea and French, et al. and Love, et al. did prospective surveys of acute upper and lower respiratory disease in families residing in different areas of pollution exposure in Chicago and New York City.[82,83] Participating families (about 600 in Chicago and about 1000 in New York City) were called biweekly by trained interviewers using a standardized questionnaire asking about the presence of illness, fever, respiratory symptoms, restricted activity, ear infection diagnosed by a physician, and other physician consultation visits. Upper respiratory disease was classified as any one of the following: cough (dry, nonproductive), head cold, sore throat, sinus or postnasal drip, or runny nose. Lower respiratory illnesses were limited to chest colds with a persistent cough, croup, bronchitis or pneumonia. Excess upper respiratory disease was found in all residentially stable family segments in Chicago and in school and preschool children in New York. All family segments in the high pollution communities in both New York and Chicago, except for children below nursery school age in Chicago, had increased acute lower respiratory disease. Morbidity varied with age, sex, and socioeconomic status in the expected fashion. No consistent differences were found with regard to ear infections.

Hammer, et al. studied acute lower respiratory disease retrospectively in children with differing exposures to sulfur dioxide total suspended particulates, and suspended sulfates in New York City in 1972 using a questionnaire and methodology similar to the two Western smelter studies.[24] Analyses were restricted to nonasthmatic children aged 1 to 12 years from families with three or more years residence duration in the community (1134 black children and 6625 white children). Children living in Queens, the Bronx, and Sheepshead Bay experienced higher air pollution levels than those living in Riverside. Rates of "any lower respiratory disease" (a combined category), croup, bronchitis, and chest infections other than croup, bronchitis and pneumonia were significantly higher among black and white children residing in the higher pollution exposure communities. Conversely, pneumonia and hospitalization were significantly higher only among white children in the low exposure community but the absolute rates were low for both conditions in all communities. Morbidity excesses in the high exposure communities could not be explained by differences in family size and composition, crowding, nor indoor air pollution from parental cigarette smoking habits or gas stoves or gas space heaters. Furthermore, morbidity rates within the three higher exposure communities were comparable and showed only infrequent and inconsistent statistical differences. This is the first reported study of the effects of air pollution on exposed black children and they too showed morbidity excesses. This fact strengthens the link between air pollution and lower respiratory disease in children since one would expect socioeconomically similar children to be affected by air pollution independent of their color or ethnic group. Most recently, Hammer studied about 4200 black and 5200 white children in two southeastern cities with differing exposures to total suspended particulate matter and suspended sulfates, but low exposures to sulfur dioxide.[22] Significant increases in "any lower respiratory disease", croup, bronchitis, pneumonia, and hospitalization for any of these illnesses were found for both black and white children living in Birmingham, the higher particulate exposure community.

Air Pollution and Acute Lower Respiratory Disease: Asthmatic Children

Only three studies have been able to look at the frequency of acute lower respiratory disease in children with a history of asthma exposed to different levels of air pollution. This is in part due to the relatively low prevalence of asthma in the population (about 1-5%). In a study of four Utah communities involving 7763 nonasthmatic children and 475 asthmatic children, asthmatic children in the high pollution communities experienced significantly higher rates of croup. Asthmatics exposed to higher sulfate levels tended to report more total respiratory illness, pneumonia, bronchitis, and hospitalizations.[12] In a study of 4305 nonasthmatic and 290 asthmatic children in five Rocky Mountain communities, the exposed asthmatic children reported more total respiratory illness, croup and bronchitis than their unexposed counterparts.[13]

In a study of over 10,000 children living in two southeastern U.S. cities and exposed to elevated levels of total suspended particulate matter and suspended sulfates, but rather low levels of sulfur dioxide, morbidity reporting in black (156) and white (338) asthmatic children was inconsistent with regard to pollution exposure.[35] Morbidity rates were higher in Birmingham, the higher particulate exposure city, in half of the comparisons studied. Croup was significantly increased in Birmingham, although the converse was true for bronchitis ($0.10>p>0.05$) among Charlotte blacks. Exposed children in the Utah and Rocky Mountain studies were more likely to have been exposed to frequent fumigations, acid aerosols and airborne trace metals which could explain why excess morbidity was found in those two studies, but not the latter. Although all three studies suggest an increased risk of acute lower respiratory disease for exposed asthmatic children further studies will be required to fully clarify this relationship.

Air Pollution and the Incidence and Frequency of Childhood Asthma

Zweiman, et al. reviewed the effects of air pollution on asthma in 1972 without regard to the age of the study subjects.[19] In the classical smog episodes of Donora, Pennsylvania and London, England, a significantly higher proportion of asthmatics were affected when compared to nonasthmatics.[17,18] Glasser, et al. and Chiramonte, et al. reported increased asthmatic episodes in both older adults and children during an air pollution episode in New York City with the peak flare-ups treated in emergency rooms on the third day of the episode.[31,32]

Zeidberg, et al. followed 84 asthmatic patients clinically for one year in Nashville, Tennessee.[86] Of these, 35 were children, 25 of whom were male and 21 of whom were white. Monthly sulfur dioxide and particulates were measured. In adults, but not children, the attack rates varied directly with the level of sulfation on their residential environment. Lewis, et al. studied Charity Hospital emergency clinic admissions for asthma (1960-61) and between 50 and 85 asthmatic residents in one census tract in New Orleans.[87] Virtually all subjects were over twelve years of age and the analyses were only for blacks since they comprised about 90% of the Charity Hospital emergency clinic population. Hi-vol air monitoring for particulate matter was done in one station near census tract 130 in 1969 and 1961. Microscopy was also done on the particulate matter. The daily number of asthmatic patients admitted to the clinic correlated with "poor combustion particles with associated silica." No statistically significant relationship between measured particles and asthma attacks in census tract 130 was observed. This study used crude air pollution measurements and was restricted to adults, but it did not find a positive relationship between the air pollution and the frequency of asthma attacks.

Girsh, et al. studied weather, air pollution, and bronchial asthma in about 1400 asthmatic children at St. Christopher's Hospital for Children in Philadelphia.[88] They found a threefold increase of bronchial asthma during days of "noteworthy high air pollution." No specific pollutants or concentrations thereof are given in this report. Sultz, et al. studied the relation of air pollution exposure to asthma and eczema in hospitalized children under 15 years of age in Erie County, New York State.[21] Air pollutant measurements were obtained from several monitoring stations. A striking positive relationship between standardized morbidity ratios for hospitalization for asthma and eczema and four levels of air pollution exposure was found. The relationship for air pollution was most striking for hospitalized males under 5 years of age with asthma or eczema. Understandably, no community incidence rates of asthma or eczema in children were obtained. The sex ratio for asthma was almost 2 to 1 for males and hospitalization for asthma was more than three times higher in children under 5 when compared to those age 5 to 15 years. It is noteworthy that the affected children in the study of Chiaramonte, et al. had a high incidence of extrinsic allergic manifestations.[85]

Two studies using similar methodologies have been reported recently. Persons with known asthma reported daily attack rates via weekly diaries in Utah (211 persons) and New York City (148 persons) for about 6 months.[89,90] In both studies, about half the study subjects were 16 years old or less. Asthma attack rates in both studies correlated negatively with ambient temperature and to a less or extent, positively with ambient total suspended particulate matter and suspended sulfates. Because the panelists were not analyzed by age, it was not possible to tell if the relationships for asthma in children to temperature and air pollution were less than, equal to, or greater than those in the adults.

Goldstein and Block[91] studied emergency room visits for asthma and air pollution in two inner city areas in New York City, viz. Harlem in Manhatten and Bedford-Stuyvesant in Brooklyn. Daily emergency room visits for asthma averaged 22 at Harlem Hospital and 59 for the two Brooklyn hospitals combined. The percent of pediatric visits in each hospital was as follows: Harlem Hospital Center-18%, Kings County Hospital-46%, Cumberland Hospital-not given. There was a strong relationship between daily visits for asthma and the first cold spells of the fall season in both areas. Daily visits for asthma correlated with daily sulfur dioxide levels in Brooklyn but not in Harlem. No data are given on the socioeconomic status, race, or the ethnic group of the persons making the visits. The relationship between daily asthma visits and daily sulfur dioxide was consistent for those under 13 years of age and those 13 years and over, but was more pronounced in the younger group.

Little information is available on the prevalence of childhood asthma in communities with high and low air pollution levels. No consistent trends of asthma prevalence and air pollution were found in the three United States surveys even though the expected excesses by sex among male children and female adults were observed.[12,13,35] A recent report from Japan did find an increased prevalence of asthma and related allergic conditions in a high pollution community which apparently had exposure to petrochemical wastes as well as sulfur dioxide and total suspended particulate matter.[92]

Summary and Conclusions

Studies from several countries clearly associate excess lower respiratory tract morbidity in nonasthmatic children with exposure to sulfur oxides and particulate matter. Most studies were retrospective, using a questionnaire and several clinically examined children at some point in time. The more recent studies noted duration of residence, and a history of asthma in the children, as well. Different investigations looked at different aged children, and the period of recall for the questionnaire, as well as its specific format, varied somewhat from study to study. The use of slightly different methods to find the association between acute lower respiratory disease and air pollution strengthens, rather than weakens, the credibility of the relationship.

The picture is relatively clear for air pollution and acute upper respiratory tract disease, as well. Two U.S. studies found a relationship between air pollution and acute upper respiratory tract disease, whereas a British and a Russian study actually found the association for chronic upper respiratory tract illnesses.[7,10,82,83] None of the studies obtained an extensive allergy history from the children, and of course the pollutant types and concentrations varied in the four studies. Nevertheless, an attractive hypothesis is that air pollution increases acute upper respiratory infections in all children and it also increases chronic upper respiratory tract disease and symptoms in children with a history of asthma or allergic conditions. This hypothesis is still conjectural, but biologically plausible.

Relatively few studies have been concerned with air pollution and asthmatic children, per se. Asthma prevalence in childhood is low and it is epidemiologically distinct from adult asthma.[15] Moreover, the disease "asthma" is markedly heterogeneous in its manifestations in children ranging from a few attacks for a short time to chronic severe, crippling, respiratory symptoms.[34,52,68-70] Aside from the difficulties in assessing air pollution exposure, almost none of the studies of asthma and air pollution classified their study subjects by severity of asthma. Moreover, although several studies measured daily temperature, other known determinants of asthmatic attacks such as pollens, dusts, grasses, and other allergens were not measured (largely due to the technical difficulties of monitoring such exposures in the field). If one assumes that only more severe cases of childhood asthma are hospitalized, then the work of Sultz, et al. showed a clear gradient with increasing pollution exposure.[21] What is needed are well-planned studies using good air-pollution measurements and careful selection and classification of asthmatic children. However, the literature to date suggests both acute and chronic effects of air pollution in asthmatic children as well as those without a history of asthma.

APPENDIX B

DATA ANALYSIS AND HYPOTHESIS TESTING

Morbidity conditions were analyzed in a saturated analysis of variance (ANOVA) form of a linear model for categorical data, viz, four main effects [city/pollution (P), age (A), sex (S), education of head of household (E)], and all possible interactions (six first-order, four second-order, and one third-order). For each morbidity condition analyzed in the model, age (three categories: 1-14 years, 5-8 years, 9-12 years), sex (two categories: F - female; M - male), and education of the head of the household (two categories: <HS - less than high school, >HS - high school or more) as an index of socioeconomic status (SES) were considered as intervening variables, and city/pollution (two categories: C - Charlotte, B - Birmingham) as the independent variable of primary interst. When statistically significant interaction terms involving the independent variable, "city/pollution" were found, they were explored further to distinguish the "city/pollution" effect from those of age, sex, and socioeconomic status.

Subsequent models to account for the significant "city/pollution" interaction were sought in a simple, logically consistent fashion. In brief, for the morbidity conditions in which only one first order interaction term involving "city/pollution" was significant, viz. city x age, city x sex, or city x SES, a subsequent reduced model was fit with the four main effects, the city effect being fit within the levels of the non-city variable (age, sex, or SES) causing the interaction. For example, to account for a significant city x sex interaction in the saturated model, the terms in the subsequent reduced model would be as follows: city for females, city for males, age, sex, and SES.

In one case all three first order "city" interaction terms were significant. When more than one significant first order interaction involving "city" occurs in the saturated model, it is not possible to fit the "city" effect within the levels of more than one of the non-city variables because it would create a singularity in the design matrix. Therefore, city, age, and SES were tested separately by sex. Sex, rather than age or SES, was chosen because of the well-known biological differences in infectious disease experiences between males and females. A significant city x age x SES interaction occurred three times and this was accounted for by the following model: city, age, sex, and SES for age within city. The resulting reduced ANOVA models and their corresponding model adjusted rates are presented in detail in the following section. However, the verbal summary of the findings is brief as they are discussed in detail in the main text of the report.

For both races, a total of seventeen reported morbidity conditions were analyzed (two races x five morbidity conditions x two number of episode categories less the three following conditions which were not statistically tested). Two or more episodes of hospitalization among both races and two or more episodes of pneumonia among whites were not tested statistically because their rates were too low to obtain reliable estimates from the model. Statistically significant interactions involving the "city/pollution" term were found in ten of the seventeen morbidity conditions tested, (this includes significance level d (0.10>p>0.05) but excludes third order interactions).

Chi-square and significance levels for the reduced ANOVA models used to account for the significant "city" interactions in black children are summarized in Table B-1 along with those for the "city" term from the saturated model for comparison (cf. Table 3). No significant interactions involving the "city/pollution" term were found for three of the nine morbidity conditions tested. A significant first order interaction term involving "city/pollution" was found in four cases. All three first order "city" interaction terms were significant in one case as was a city x age x SES interaction in another. For black children, model adjusted rates from the reduced models are presented in Table B-2 for those morbidity conditions with one significant first order "city" interaction and in Table B-3 for the two morbidity conditions with either three significant first order "city" interactions or a significant second order interaction.

For white children, chi-square and significance levels for reduced models used to account for the significant "city" interactions are summarized in Table B-4 along with those for the "city" term from the saturated model for comparison (cf. Table 4). No significant "city" interactions were found for four of the eight morbidity conditions tested. (Third order interactions did approach statistical significance (0.10>p>0.05) in three cases but were ignored because of the general difficulty of interpreting them.) A significant first order interaction term involving "city/pollution" was found in two cases and a significant city x age x SES interaction was found in the two others. Model adjusted rates from the reduced models for white children are presented in Table B-5 for the two morbidity conditions with a significant first order "city" interaction and in Table B-6 for the two morbidity conditions with a significant second order "city" interaction.

Morbidity conditions were analyzed initially in a saturated analysis of variance form (four main effects and all possible interactions) of a linear model for categorical data except for three conditions in which the reported rates were too low. Statistically significant interactions involving the independent variable, "city/pollution", were found in 6 of 9 morbidity conditions in black children and 4 of 8 conditions in white children (Table B-7). Thus statistically significant "city/pollution" interactions were found in just over half (10/17) of all tested morbidity conditions. Models to account for the interactions were sought by reducing the saturated model to the four main effects (city/pollution, age, sex, SES) and fitting the city effect within the non-city variable whenever possible. Six of the ten cases involved only one significant first order "city/pollution" interaction each and reduced models were fit with the four main effects, the city effect being fit within the levels of the non-city variable (age, sex, or SES). Significant city x age x SES interactions were found in three of the 10 cases and were accounted for by the following reduced model: city, age, sex, SES for age within city. In one case, all three first order "city/pollution" interactions were significant and a reduced model which analyzed city, age, and SES for each sex was used to account for the interaction. When the city/pollution effect was distinguished from the effects of age, sex, and socioeconomic status in the analysis of variance, morbidity rates often were significantly higher in Birmingham and in no case was the converse true.

TABLE B-1. CHI-SQUARE AND SIGNIFICANCE LEVELS FOR FACTORS AFFECTING LOWER RESPIRATORY DISEASE AS DETERMINED BY A REDUCED LINEAR MODEL FOR CATEGORICAL DATA: BLACK CHILDREN WITH THREE OR MORE YEARS OF COMMUNITY RESIDENCE

Effect (d.f.)	ANY LRD			CROUP	
	>1	≥2		>1	≥2
Saturated Model City/Pollution(1)	0.79	0.09		0.02	0.01
Reduced Model City/Pollution(1)	F-7.15(1)^b M-0.24(1)	Female 2.39(1)	Male 2.43(1)	<HS-0.86(1)^c ≥HS-4.52(1)	1-4 2.70(1)$_b$ 5-8 7.40(1) 9-12 0.53(1)
Age (2)	30.01^a	C-2.93 B-2.71	10.69^b 4.21	18.42^a	3.92
Sex(1)	0.73	-	-	0.13	0.51
SES	3.11(1)^d	C-0.02(1)$_b$ B-5.07(1)^b	0.03(1)$_b$ 5.44(1)^b	3.99(1)^c	0.02(1)
Charlotte Age 1-4(1) Age 5-8(1) Age 9-12(1)					
Birmingham Age 1-4(1) Age 5-8(1) Age 9-12(1)					
Fit of Model	16.87(17)	10.38(9)		15.22(17)	14.56(16)

a-p≤0.001; b-p≤0.01; c-p≤0.05; d-0.10>p>0.05 For each term in the model, the probability for a two-tailed test of statistical significance

(continued)

TABLE B-1. (continued)

Effect (d.f.)	BRONCHITIS		PNEUMONIA		HOSPITALIZATION	
	>1	≥2	>1	≥2	>1	≥2
Saturated Model City/Pollution(1)	0.60	0.71	8.12^b	0.99	2.50	
Reduced Model City/Pollution(1)	*	*	<HS-$3.38(1)^d$ ≥HS-$10.59(1)^a$	*	4.54^c	Rate
Age(2)			12.88^b		12.05^b	too
Sex (1)			0.18		0.76	low
SES			0.01(1)			to
Charlotte						fit
Age 1-4(1)					7.56^b	
Age 5-8(1)					1.43	model.
Age 9-12(1)					0.12	
Birmingham						
Age 1-4(1)					1.78	
Age 5-8(1)					9.05^b	
Age 9-12(1)					10.24^b	
Fit of Model			19.98(17)		18.24(13)	

* Saturated model adequate

a-p≤0.001
b-p≤0.01
c-p≤0.05
d-0.10>p>0.05

For each term in the model, the probability for a two-tailed test of statistical significance.

TABLE B-2. FOUR YEAR FREQUENCY OF EACH MORBIDITY CONDITION BY NUMBER OF EPISODES AND COMMUNITY: MODEL ADJUSTED RATES FOR BLACK CHILDREN AGED 1 TO 12 YEARS

Effect	ANY LRD			CROUP				
	>1		>2	>1		>2		
Saturated Model								
Charlotte	18.7%		10.1%	7.0%		2.4%		
Birmingham	20.0%		10.4%	7.1%		2.5%		
Reduced Model								
	Female	Male		<HS	>HS	1-4	5-8	9-12
Charlotte	16.6%	18.4%	-[a]	7.4%	5.7%	3.6%	1.6%	2.3%
Birmingham	21.0%	19.2%		6.5%	8.2%	1.4%	3.5%	2.7%

a-Three first order "city" interactions, cf. Table B-3 for model-adjusted rates

(continued)

TABLE B-2. (continued)

Effect	BRONCHITIS >1	>2	PNEUMONIA >1		>2	HOSPITALIZATION >1	>2
Saturated Model							
Charlotte	9.2%	3.6%	9.5%		4.1%	2.6%	Rate too low to fit model.
Birmingham	8.4%	3.0%	12.8%		4.9%	3.6%	
Reduced Model							
			>HS	>HS			
Charlotte	*	*	9.5%	7.8%	*	_b	
Birmingham			11.8%	13.4%			

*Saturated model adequate, i.e. no significant interactions involving the "city/pollution" term.

b-City x age x SES interaction, cf. Table B-3 for model-adjusted rates from reduced model.

TABLE B-3. FOUR YEAR FREQUENCY OF "ANY LOWER RESPIRATORY DISEASE" AND HOSPITALIZATION: MODEL ADJUSTED RATES FOR BLACK CHILDREN AGED 1 TO 12 YEARS

Age	SES	Two or More Episodes of "Any Lower Respiratory Disease"				One or More Episodes of Hospitalization	
		Females		Males			
		Charlotte	Birmingham	Charlotte	Birmingham	Charlotte	Birmingham
1-4 Years	<HS	11.7%	9.4%	18.6%	6.4%	6.9%	3.5%
	>HS	12.0%	13.4%	18.3%	10.1%	1.3%	6.5%
5-8 Years	<HS	7.0%	10.3%	7.3%	8.7%	1.9%	3.6%
	>HS	7.2%	14.2%	7.0%	12.4%	0.9%	0.9%
9-12 Years	<HS	7.0%	7.3%	7.8%	6.4%	1.0%	3.3%
	>HS	7.3%	11.2%	7.5%	10.1%	1.3%	0.7%

TABLE B-4. CHI-SQUARE AND SIGNIFICANCE LEVELS FOR FACTORS AFFECTING LOWER RESPIRATORY DISEASE AS DETERMINED BY A REDUCED LINEAR MODEL FOR CATEGORICAL DATA: WHITE CHILDREN WITH THREE OR MORE YEARS OF COMMUNITY RESIDENCE

Effect (d.f.)	Any LRD		Croup	
	≥1	≥2	≥1	≥2
Saturated Model				
City/Pollution(1)	9.91^b	10.99^a	0.74	4.59^c
Reduced Model				
City/Pollution(1)	*	*	3.97^c	*
Age(2)			27.03^a	
Sex(1)			0.09	
SES				
Charlotte				
Age 1-4(1)			0.18	
Age 5-8(1)			4.30^c	
Age 9-12(1)			0.22	
Birmingham				
Age 1-4(1)			6.80^b	
Age 5-8(1)			0.96	
Age 9-12(1)			0.07	
Fit of Model			18.81(13)	

Continued

*Saturated model adequate
a-$p \leq 0.001$
b-$p \leq 0.01$
c-$p \leq 0.05$
d-$0.10 > 0.05$

TABLE B-4. (Continued)

Effect (d.f.)	Bronchitis		Pneumonia		Hospitalization	
	≥1	≥2	≥1	≥2	≥1	≥2
Saturated Model						
City/Pollution(1)	11.99^a	24.18^a	3.43^d		5.53^c	
Reduced Model						
City/Pollution(1)	F- $2.76(1)^d$ M-$16.81(1)^a$	*	5.05^c	Rate too low to fit model	<HS- 2.46(1) ≥HS-$12.30(1)^a$	Rate too low to fit model
Age(2)	40.86^a		9.66^b		36.82^a	
Sex(1)	4.17^c		0.37		0.51	
SES	$18.23(1)^a$				$8.51(1)^b$	
Charlotte						
Age 1-4(1)			0.32			
Age 5-8(1)			6.34^b			
Age 9-12(1)			<0.01			
Birmingham						
Age 1-4(1)			0.05			
Age 5-8(1)			1.20			
Age 9-12(1)			6.21^b			
Fit of Model	13.12(17)		13.96(13)		13.79(17)	

*Saturated model adequate
a-$p \leq 0.001$
b-$p \leq 0.01$
c-$p \leq 0.05$
d-$0.10 > 0.05$

TABLE B-5. FOUR YEAR FREQUENCY OF EACH MORBIDITY CONDITION BY NUMBER OF EPISODES AND COMMUNITY: MODEL ADJUSTED RATES FOR WHITE CHILDREN AGED 1 to 12 YEARS

Effect	Any LRD		Croup		Bronchitis		Pneumonia		Hospitalization	
	≥1	≥2	≥1	≥2	≥1	≥2	≥1	≥2	≥1	≥2
Saturated Model										
Charlotte	28.7%	16.2%	13.5%	5.6%	19.3%	8.3%	6.8%	Rate	3.0%	Rate
Birmingham	33.6%	20.3%	14.5%	7.4%	24.2%	13.6%	8.5%	too	4.6%	too
								low		low
Reduced Model					Female Male			to	<HS >HS	to
Charlotte					20.3% 18.2%			fit	3.1% 2.5%	fit
Birmingham	*	*	**		22.9% 25.0%	*	**	model	4.1% 4.8%	model

*Saturated model adequate, i.e. no significant interactions involving the "city/pollution" term.
**City x age x SES interaction.

TABLE B-6. FOUR YEAR FREQUENCY OF CROUP AND PNEUMONIA: MODEL ADJUSTED RATES FOR WHITE CHILDREN AGED 1 TO 12 YEARS

Age	SES	One or More Episodes of Croup		One or More Episodes of Pneumonia	
		Charlotte	Birmingham	Charlotte	Birmingham
1-4 Years	<HS	14.2%	11.8%	7.4%	10.2%
	>HS	15.2%	22.1%	8.8%	9.4%
5-8 Years	<HS	12.0%	17.3%	9.1%	7.1%
	>HS	16.2%	14.7%	5.5%	9.2%
9-12 Years	<HS	9.2%	11.8%	4.7%	8.6%
	>HS	9.9%	11.2%	4.9%	4.6%

TABLE B-7. SUMMARY OF STATISTICALLY SIGNIFICANT[a] INTERACTIONS IN WHICH THE "CITY/POLLUTION" EFFECT WAS INVOLVED, BY MORBIDITY CONDITION AND RACE

Morbidity Condition	Number of Episodes	Black Children	White Children
"Any Lower Respiratory Disease"	≥1	C* x S[b]	None
	≥2	C x A,C x S,C x E	None
Croup	≥1	C x E[b]	C x A x E
	≥2	C x A	None
Bronchitis	≥1	None	C x S[b]
	≥2	None	None
Pneumonia	≥1	C x E[b]	C x A x E
	≥2	None	-[c]
Hospitalization	≥1	C x A x E	C x E
	≥2	-[c]	-[c]

a-$p \leq 0.05$

b-$0.10>p>0.05$

c-Rate too low to fit model

*Explanation of abbreviations:

C-City/Pollution
A-Age
S-Sex
E-Education, head of household

APPENDIX C

VALIDITY AND RELIABILITY OF DISEASE REPORTING

The validity and reliability of disease reporting by means of the questionnaire was not determined in this study. However, there is an abundant amount of direct and indirect evidence from this and other studies which lend credence to the observed results. In two other studies (Utah and Rocky Mountain) using this questionnaire, 15% samples of children whose parents reported them sick or well for bronchitis were taken, and the parents' reports were compared with information in physicians' records of the same children.[12,13] Rather than sensitivity and specificity, this is akin to what Vecchio called the "positive" and "negative" predictive values (PVpos, PVneg) of a single diagnostic test, i.e. the proportion of true positives among those who test positive and the proportion of true negatives among those who test negative, respectively.[93] The PVpos and PVneg vary with the true prevalence of the attribute as well as the specificity and sensitivity of the test (or questionnaire). Theoretical expected PVpos and PVneg have been calculated for varying sensitivities, specificities and true prevalence (Table C-1). For example, for a true prevalence of 10% and a sensitivity and specificity of 50%, the expected PVpos and PVneg are 10% and 90% respectively; they are 20% and 80% for a true prevalence of 20%. If the sensitivity and specificity are both 90%, the PVpos and PVneg become 50% and 99% for a true prevalence of 10%, and 69% and 97% for a true prevalence of 20%. Alterations in true prevalence, sensitivity, and specificity have a greater effect upon PVpos than upon PVneg.

In the Utah study, the overall frequency of bronchitis was about 20%, the PVpos about 75% and the PVneg about 90%. In the Rocky Mountain study, the overall frequency of bronchitis was about 15%, the PVpos about 75% and the PVneg about 85%. Hence, the PVpos and PVneg of the two studies were quite similar. From the expected values in Table C-1, it appears that the negative predictive values were in accord with expectation or a bit low and that the positive predictive values were too high, if anything, unless the sensitivity of the questionnaire is 90% or better. No further conclusions can be made regarding the sensitivity and specificity of the questionnaire from these data until further information is available. Sampling for PVpos and PVneg in the Utah and Rocky Mountain studies was not restricted by age, education of the head of the household, history of asthma or duration of residence in the community (so that in several cases, the physician listed on the questionnaire was not the child's current physician).

The specificity and sensitivity of this questionnaire could be determined by querying a sample of parents whose children have been cared for by a known group of physicians or health clinic for some length of time. Of course, such results would not directly apply to this study. However, they would provide some quantitative estimates and would be especially useful if families from a broad range of social classes were included and children were also sampled with regard to age, history of asthma and duration of residence in the community (to assure that the illness was in fact treated in their current community of residence).

Our rates for "any lower respiratory disease" were about 286 and 336/1000 white, nonasthmatic children per four years in Charlotte and Birmingham, figures remarkably in accord with the rate of 85/1000 children (aged 1-12 years) per year found by Glezen and Denny in a prospective study with relatively frequent medical attention for participating families.[29] Our data are in accord with U.S. National Health Survey data which show that although chronic bronchitis prevalence varies inversely with the education of the head of the family for persons aged 17 years and over, chronic bronchitis prevalence actually increases with parental education for children under seventeen years of age.[59] Children with a history of asthma would be expected to have an increased risk of lower respiratory disease. Not only was an increased risk found in this study, but it generally was intermediate among children with inactive asthma and highest among those with active asthma.

Asthma prevalence differed by sex among children and adults as expected. The relation of cigarette smoking to education of the head of the household, age, and sex, and the relationship of chronic respiratory disease symptom prevalenc to smoking and a history of occupational exposure in parents of these children also conformed to expectation (results not reported in this report). Return rates were excellent in both cities and missing information was minimal. Black-white morbidity differences were found in both communities and were confirmed in a recent report which utilized virtually the same questionnaire.[24] Yet similar patterns of morbidity with respect to age, education of the head of the household, and a history of asthma were found in both black and white children.

In summary, the evidence for the validity and reliability of the questionnaire is as follows: empirical PVpos and PVneg in accord with expectation (in two studies) given the uncertainties of these two statistics; the comparability of the rates in this study to those found in a prospective study, the agreement with U.S. National Health Survey data regarding bronchitis in children and education of the head of the household; the increased risk of lower respiratory disease following a gradient among nonasthmatic, inactive, and asthmatic children; the expected relationship of asthma prevalence to sex among children and adults of both races; the expected relationships among cigarette smoking, age, sex, history of occupational exposure, and chronic bronchitis symptom prevalence in the parents of these children; the excellent return rates and low missing information rates, and the consistent black-white morbidity differences which have been confirmed in another study. All of these facts argue strongly for the reliability and the validity of the questionnaire.

TABLE C-1. THEORETICAL EXPECTED POSITIVE AND NEGATIVE PREDICTIVE VALUES UNDER VARYING SENSITIVITY AND SPECIFICITY AND A TRUE PREVALENCE (p_t) OF 10% or 20%.

a. Sensitivity and Specificity Variable

Sensitivity = Specificity	PV Positive		PV Negative	
	p_t = 10%	p_t = 20%	p_t = 10%	p_t = 20%
50%	10%	20%	90%	80%
75%	25%	43%	96%	92%
90%	50%	69%	99%	97%

b. Sensitivity = 75%, Specificity Variable

Sensitivity = 75% Specificity	PV Positive		PV Negative	
	p_t = 10%	p_t = 20%	p_t = 10%	p_t = 20%
50%	14%	27%	95%	89%
75%	25%	43%	96%	92%
90%	45%	65%	97%	94%

c. Sensitivity Variable, Specificity = 75%

Specificity = 75% Sensitivity	PV Positive		PV Negative	
	p_t = 10%	p_t = 20%	p_t = 10%	p_t = 20%
50%	18%	33%	93%	86%
75%	25%	43%	96%	92%
90%	29%	47%	99%	97%

APPENDIX D

OBSERVED MORBIDITY RATES

Actual reported morbidity rates by age, sex, and education of the head of the household are given here for reference (Tables D-1 through D-10).

TABLE D-1. "ANY LOWER RESPIRATORY DISEASE": REPORTED FOUR YEAR FREQUENCY AMONG BLACK, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY RESIDENCE

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	21.7(13/60)	16.8(32/190)	11.7(28/239)	14.9(73/489)
		Birmingham	20.5(24/117)	20.8(52/250)	17.5(57/325)	19.2(133/692)
	≥HS	Charlotte	24.3(18/74)	17.7(25/141)	11.9(19/159)	16.6(62/374)
		Birmingham	31.5(28/89)	19.6(45/230)	18.9(43/228)	21.2(116/547)
Male	<HS	Charlotte	25.4(18/71)	13.1(21/160)	14.3(29/203)	15.7(68/434)
		Birmingham	20.0(23/115)	16.7(46/276)	13.3(43/323)	15.7(112/714)
	≥HS	Charlotte	38.8(19/49)	17.6(24/136)	13.1(23/175)	18.3(66/360)
		Birmingham	23.3(21/90)	24.7(56/227)	13.7(31/226)	19.9(108/543)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	10.0(6/60)	6.3(12/190)	7.5(18/239)	7.4(36/489)
		Birmingham	6.8(8/117)	10.8(27/250)	7.7(25/325)	8.7(60/692)
	≥HS	Charlotte	12.2(9/74)	7.8(11/141)	6.3(10/159)	8.0(30/374)
		Birmingham	20.2(18/89)	13.0(30/230)	10.1(23/228)	13.0(71/547)
Male	<HS	Charlotte	18.3(13/71)	6.9(11/160)	8.9(18/203)	9.7(42/434)
		Birmingham	5.2(6/115)	8.3(23/276)	6.2(20/323)	6.9(49/714)
	≥HS	Charlotte	22.4(11/49)	8.1(11/136)	6.9(12/175)	9.4(34/360)
		Birmingham	15.6(14/90)	13.7(31/227)	7.5(17/226)	11.4(62/543)

TABLE D-2. CROUP: REPORTED FOUR YEAR FREQUENCY AMONG BLACK, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY RESIDENCE

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	10.0(6/60)	8.4(16/190)	2.9(7/239)	5.9(29/489)
		Birmingham	6.8(8/117)	6.4(16/250)	6.2(20/325)	6.4(44/692)
	≥HS	Charlotte	8.1(6/74)	5.7(8/141)	3.8(6/159)	5.3(20/374)
		Birmingham	11.2(10/89)	8.7(20/230)	8.8(20/228)	9.1(50/547)
Male	<HS	Charlotte	8.5(6/71)	7.5(12/160)	6.9(14/203)	7.4(32/434)
		Birmingham	6.1(7/115)	6.5(18/276)	2.5(8/323)	4.6(33/714)
	≥HS	Charlotte	16.3(8/49)	6.6(9/136)	5.1(9/175)	7.2(26/360)
		Birmingham	10.0(9/90)	9.7(22/227)	5.8(13/226)	8.1(44/543)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	1.7(1/60)	1.6(3/190)	1.3(3/239)	1.4(7/489)
		Birmingham	1.7(2/117)	3.6(9/250)	2.5(8/325)	2.7(19/692)
	≥HS	Charlotte	5.4(4/74)	0.0(0/141)	1.3(2/159)	1.6(6/374)
		Birmingham	2.2(2/89)	2.6(6/230)	3.1(7/228)	2.7(15/547)
Male	<HS	Charlotte	5.6(4/71)	1.3(2/160)	2.5(5/203)	2.5(11/434)
		Birmingham	0.9(1/115)	2.9(8/276)	0.9(3/323)	1.7(12/714)
	≥HS	Charlotte	6.1(3/49)	1.5(2/136)	0.0(0/175)	1.4(5/360)
		Birmingham	3.3(3/90)	3.1(7/227)	3.1(7/226)	3.1(17/543)

TABLE D-3. BRONCHITIS: REPORTED FOUR YEAR FREQUENCY AMONG BLACK, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY RESIDENCE

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	8.3(5/60)	5.8(11/190)	6.7(16/239)	6.5(32/489)
		Birmingham	6.8(8/117)	7.6(19/250)	6.8(22/325)	7.1(49/692)
	>HS	Charlotte	16.2(12/74)	9.2(13/141)	8.2(13/159)	10.2(38/374)
		Birmingham	18.0(16/89)	8.7(20/230)	9.2(21/228)	10.4(57/547)
Male	<HS	Charlotte	15.5(11/71)	6.9(11/160)	5.9(12/203)	7.8(34/434)
		Birmingham	4.3(5/115)	7.2(20/276)	5.0(16/323)	5.7(41/714)
	>HS	Charlotte	14.3(7/49)	8.8(12/136)	4.6(8/175)	7.5(27/360)
		Birmingham	12.2(11/90)	7.9(18/227)	6.6(15/226)	8.1(44/543)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	0.0(0/60)	1.6(3/190)	2.9(7/239)	2.0(10/489)
		Birmingham	0.9(1/117)	3.6(9/250)	2.2(7/325)	2.5(17/692)
	>HS	Charlotte	8.1(6/74)	2.8(4/141)	3.1(5/159)	4.0(15/374)
		Birmingham	6.7(6/89)	1.7(4/230)	3.9(9/228)	3.5(19/547)
Male	<HS	Charlotte	9.9(7/71)	2.5(4/160)	0.5(1/203)	2.8(12/434)
		Birmingham	2.6(3/115)	3.6(10/276)	1.5(5/323)	2.5(18/714)
	>HS	Charlotte	6.1(3/49)	2.9(4/136)	2.3(4/175)	3.1(11/360)
		Birmingham	4.4(4/90)	3.1(7/227)	1.8(4/226)	2.8(15/543)

TABLE D-4. PNEUMONIA: REPORTED FOUR YEAR FREQUENCY AMONG BLACK, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY RESIDENCE

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	13.3(8/60)	8.9(17/190)	6.7(16/239)	8.4(41/489)
		Birmingham	8.5(10/117)	14.8(37/250)	11.7(38/325)	12.3(85/692)
	≥HS	Charlotte	10.8(8/74)	7.1(10/141)	3.8(6/159)	6.4(24/374)
		Birmingham	18.0(16/89)	13.5(31/230)	11.0(25/228)	13.2(72/547)
Male	<HS	Charlotte	15.5(11/71)	7.5(12/160)	8.9(18/203)	9.4(41/434)
		Birmingham	13.9(16/115)	9.8(27/276)	9.9(32/323)	10.5(75/714)
	≥HS	Charlotte	16.3(8/49)	9.6(13/136)	6.9(12/175)	9.2(33/360)
		Birmingham	18.9(17/90)	16.3(37/227)	8.4(19/226)	13.4(73/543)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	5.0(3/60)	3.2(6/190)	4.2(10/239)	3.9(19/489)
		Birmingham	4.3(5/117)	5.2(13/250)	4.0(13/325)	4.5(31/692)
	≥HS	Charlotte	5.4(4/74)	3.5(5/141)	1.9(3/159)	3.2(12/374)
		Birmingham	6.7(6/89)	6.1(14/230)	5.3(12/228)	5.9(32/547)
Male	<HS	Charlotte	5.6(4/71)	2.5(4/160)	3.4(7/203)	3.5(15/434)
		Birmingham	2.6(3/115)	4.0(11/276)	3.7(12/323)	3.6(26/714)
	≥HS	Charlotte	8.2(4/49)	2.9(4/136)	4.0(7/175)	4.2(15/360)
		Birmingham	4.4(4/90)	7.0(16/227)	5.3(12/226)	5.9(32/543)

TABLE D-5. HOSPITALIZATION: REPORTED FOUR YEAR FREQUENCY AMONG BLACK, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY RESIDENCE

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	6.7(4/60)	1.6(3/190)	1.3(3/239)	2.0(10/489)
		Birmingham	4.3(5/117)	3.6(9/250)	2.8(9/325)	3.3(23/692)
	≥HS	Charlotte	0.0(0/74)	0.0(0/141)	1.3(2/159)	0.5(2/374)
		Birmingham	7.9(7/89)	0.4(1/230)	3.5(8/228)	2.9(16/547)
Male	<HS	Charlotte	5.6(4/71)	1.9(3/160)	1.0(2/203)	2.1(9/434)
		Birmingham	3.5(4/115)	4.0(11/276)	3.4(11/323)	3.6(26/714)
	≥HS	Charlotte	4.1(2/49)	3.7(5/136)	1.7(3/175)	2.8(10/360)
		Birmingham	6.7(6/90)	4.0(9/227)	0.4(1/226)	2.9(16/543)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	0.0(0/60)	0.5(1/190)	1.3(3/239)	0.8(4/489)
		Birmingham	0.0(0/117)	1.2(3/250)	0.3(1/324)	0.6(4/692)
	≥HS	Charlotte	0.0(0/74)	0.0(0/141)	0.0(0/159)	0.0(0/374)
		Birmingham	2.2(2/89)	0.0(0/230)	0.0(0/228)	0.4(2/547)
Male	<HS	Charlotte	1.4(1/71)	0.0(0/160)	0.5(1/203)	0.5(2/434)
		Birmingham	0.0(0/115)	0.4(1/276)	0.0(0/323)	0.1(1/714)
	≥HS	Charlotte	0.0(0/49)	0.7(1/36)	0.0(0/175)	0.3(1/360)
		Birmingham	0.0(0/90)	0.4(1/227)	0.0(3/226)	0.2(1/543)

TABLE D-6. "ANY LOWER RESPIRATORY DISEASE": REPORTED FOUR YEAR FREQUENCY AMONG WHITE, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY RESIDENCE

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	26.0(25/96)	27.4(68/248)	24.0(7/296)	25.6(164/640)
		Birmingham	32.7(16/49)	32.3(62/152)	24.0(43/179)	29.0(111/380)
	≥HS	Charlotte	33.6(37/110)	32.5(109/335)	23.2(106/457)	27.9(252/902)
		Birmingham	37.2(35/94)	35.7(87/244)	21.1(58/275)	29.4(180/613)
Male	<HS	Charlotte	40.0(36/90)	26.1(60/230)	16.1(48/298)	23.3(144/618)
		Birmingham	33.8(22/65)	34.8(55/158)	29.5(57/193)	32.2(134/416)
	≥HS	Charlotte	38.5(59/153)	32.3(121/369)	24.4(117/479)	29.7(297/1001)
		Birmingham	50.6(42/83)	40.0(92/230)	27.4(72/263)	35.8(206/576)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	16.7(16/96)	18.1(45/248)	9.8(29/296)	14.1(90/640)
		Birmingham	20.4(10/49)	23.0(35/152)	11.7(21/179)	17.4(66/380)
	≥HS	Charlotte	17.3(19/110)	17.6(59/335)	13.1(60/457)	15.3(138/902)
		Birmingham	19.1(18/94)	20.9(51/244)	16.0(44/275)	18.4(113/613)
Male	<HS	Charlotte	18.9(17/90)	15.2(35/230)	9.4(28/298)	12.9(80/618)
		Birmingham	20.0(13/65)	19.6(31/158)	21.8(42/193)	20.7(86/416)
	≥HS	Charlotte	22.9(35/153)	20.3(75/309)	14.4(69/479)	17.9(179/1001)
		Birmingham	33.7(28/83)	25.2(58/230)	16.7(44/263)	22.6(130/576)

TABLE D-7. CROUP: REPORTED FOUR YEAR FREQUENCY AMONG WHITE, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	14.6(14/96)	14.1(35/248)	10.1(30/296)	12.3(79/640)
		Birmingham	12.2(6/49)	19.1(29/152)	10.6(19/179)	14.2(54/380)
	≥HS	Charlotte	15.5(17/110)	15.8(53/335)	9.8(45/457)	12.7(115/902)
		Birmingham	13.8(13/94)	16.0(39/244)	11.6(32/275)	13.7(84/613)
Male	<HS	Charlotte	20.0(18/90)	10.0(23/230)	7.7(23/298)	10.4(64/618)
		Birmingham	7.7(5/65)	16.5(26/158)	15.0(29/193)	14.4(60/416)
	≥HS	Charlotte	19.6(30/153)	16.3(60/369)	9.4(45/479)	13.5(135/1001)
		Birmingham	30.1(25/83)	13.9(32/230)	11.8(31/263)	15.3(88/576)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	5.2(5/96)	6.0(15/248)	4.1(12/296)	5.0(32/640)
		Birmingham	8.2(4/49)	10.5(16/152)	5.6(10/179)	7.9(30/380)
	≥HS	Charlotte	5.5(6/110)	7.5(25/335)	3.1(14/457)	5.0(45/902)
		Birmingham	5.3(5/94)	10.7(26/244)	7.6(21/275)	8.5(52/613)
Male	<HS	Charlotte	8.9(8/90)	3.9(9/230)	3.7(11/298)	4.5(28/618)
		Birmingham	3.1(2/65)	8.2(13/158)	10.9(21/193)	8.7(36/416)
	≥HS	Charlotte	8.5(13/153)	7.9(29/369)	4.2(20/279)	6.2(62/1001)
		Birmingham	1.2(1/83)	6.5(15/230)	5.7(15/263)	5.4(31/576)

TABLE D-8. BRONCHITIS: REPORTED FOUR YEAR FREQUENCY AMONG WHITE, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY RESIDENCE

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	19.8(19/96)	19.0(47/248)	13.5(40/296)	16.6(106/640)
		Birmingham	18.4(9/49)	21.1(32/152)	14.0(25/179)	17.4(66/380)
	≥HS	Charlotte	22.7(25/110)	22.4(75/335)	15.8(72/457)	19.1(172/902)
		Birmingham	28.7(27/94)	26.2(64/244)	19.6(54/275)	23.7(145/613)
Male	<HS	Charlotte	27.8(25/90)	15.7(36/230)	11.4(34/298)	15.4(95/618)
		Birmingham	26.2(17/65)	24.7(39/158)	20.2(39/193)	22.8(95/416)
	≥HS	Charlotte	22.2(34/158)	23.8(88/369)	18.4(88/479)	21.0(210/1001)
		Birmingham	39.8(33/83)	32.1(74/230)	20.2(53/263)	27.8(160/576)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	6.3(6/96)	10.1(25/248)	3.7(11/296)	6.6(42/640)
		Birmingham	12.2(6/49)	13.2(20/152)	15.2(12/179)	10.0(38/380)
	≥HS	Charlotte	9.1(10/110)	9.3(31/335)	7.7(35/457)	8.4(76/902)
		Birmingham	10.6(10/94)	14.3(35/244)	12.0(33/275)	12.7(78/613)
Male	<HS	Charlotte	8.9(8/90)	7.8(18/230)	5.0(15/298)	6.6(41/618)
		Birmingham	16.9(11/65)	12.7(20/158)	11.9(23/193)	13.0(54/416)
	≥HS	Charlotte	10.5(16/153)	12.7(47/369)	10.0(48/479)	11.1(111/1001)
		Birmingham	25.3(21/83)	17.4(40/230)	11.4(30/263)	15.8(91/576)

TABLE D-9. PNEUMONIA: REPORTED FOUR YEAR FREQUENCY AMONG WHITE, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY RESIDENCE

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	10.4(10/96)	7.3(18/248)	6.8(20/296)	7.5(48/640)
		Birmingham	12.2(6/49)	6.6(10/152)	7.8(14/179)	7.9(30/380)
	≥HS	Charlotte	5.5(6/110)	3.9(13/335)	5.0(23/457)	4.7(42/902)
		Birmingham	12.8(12/94)	9.0(22/244)	4.0(11/275)	7.3(45/613)
Male	<HS	Charlotte	5.6(5/90)	9.1(21/230)	4.0(12/298)	6.1(38/618)
		Birmingham	9.2(6/65)	8.2(13/158)	8.8(17/193)	8.7(36/416)
	≥HS	Charlotte	14.4(22/153)	5.4(20/369)	5.0(24/479)	6.6(66/1001)
		Birmingham	7.2(6/83)	10.0(23/230)	4.9(13/263)	7.3(42/576)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	2.1(2/96)	2.0(5/248)	1.4(4/296)	1.7(11/640)
		Birmingham	4.1(2/49)	3.3(5/152)	2.8(5/179)	3.2(12/380)
	≥HS	Charlotte	0.9(1/110)	1.8(6/335)	2.0(9/457)	1.8(16/902)
		Birmingham	4.3(4/94)	2.5(6/244)	1.1(3/275)	2.1(13/613)
Male	<HS	Charlotte	1.1(1/90)	3.0(7/230)	1.7(5/298)	2.1(13/618)
		Birmingham	1.5(1/65)	3.8(6/158)	5.2(10/193)	4.1(17/416)
	≥HS	Charlotte	5.2(8/153)	1.6(6/369)	1.0(5/479)	1.9(19/1001)
		Birmingham	1.2(1/83)	1.7(4/230)	0.4(1/263)	1.0(6/576)

TABLE D-10. HOSPITALIZATION: REPORTED FOUR YEAR FREQUENCY AMONG WHITE, NONASTHMATIC CHILDREN WITH THREE OR MORE YEARS OF FAMILIAL COMMUNITY RESIDENCE

a. One or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	6.3(6/96)	1.6(4/248)	0.3(1/296)	1.7(11/640)
		Birmingham	2.0(1/49)	3.9(6/152)	2.2(4/179)	2.9(11/380)
	≥HS	Charlotte	5.5(6/110)	3.6(12/335)	1.1(5/457)	2.5(23/902)
		Birmingham	6.4(6/94)	6.6(16/244)	3.3(9/275)	5.1(31/613)
Male	<HS	Charlotte	4.4(4/90)	3.0(7/230)	1.0(3/298)	2.3(14/618)
		Birmingham	4.6(3/65)	3.8(6/158)	1.6(3/193)	2.9(12/416)
	>HS	Charlotte	5.9(9/153)	2.7(10/369)	1.3(6/479)	2.5(25/1001)
		Birmingham	9.6(8/83)	9.6(22/230)	2.3(6/263)	6.3(36/576)

b. Two or More Episodes

Sex	Education, Head of Household	City	1-4 Years	5-8 Years	9-12 Years	All Ages
Female	<HS	Charlotte	1.0(1/96)	0.0(0/248)	0.0(0/296)	0.2(1/640)
		Birmingham	0.0(0/49)	1.3(2/152)	0.6(1/179)	0.8(3/380)
	≥HS	Charlotte	0.9(1/110)	0.0(0/335)	0.0(0/457)	0.1(1/902)
		Birmingham	1.1(1/94)	2.0(5/244)	0.4(1/275)	1.1(7/613)
Male	<HS	Charlotte	0.0(0/90)	0.4(1/230)	0.0(0/298)	0.2(1/618)
		Birmingham	0.0(0/65)	1.3(2/158)	0.0(0/193)	0.5(2/416)
	≥HS	Charlotte	2.0(3/153)	0.5(2/369)	0.2(1/479)	0.6(6/1001)
		Birmingham	1.2(1/83)	1.3(3/230)	0.0(0/263)	0.7(4/576)

APPENDIX E

QUESTIONNAIRE USED IN STUDY

3201 0199

(CARD 1)

FORM APPROVED
Budget Bureau
No. 85-R0133

SCHOOL AND FAMILY HEALTH QUESTIONNAIRE

FAMILY SURNAME: ______________________________
(COL. 9-28)

ADDRESS: ______________________________
(COL. 29-59)

TELEPHONE: ______________________________
(COL. 60-66)

Please write on the lines above your family's surname (last name), address, and telephone number.

The information requested in this questionnaire will be held in strict confidence. It will be averaged for groups of people only.

(COL. 79-80)
0 1

I. HEALTH QUESTIONS CONCERNING FATHER AND MOTHER ONLY

(CARD 2)

NOTE: The questions in this section are to be answered for both parents by the mother or female guardian. Answer the questions in this section only for parents (or guardians) living in the home.

	MOTHER (or female guardian)		FATHER (or male guardian)	
	(COL. 9)		(COL. 10)	
	Yes	No	Yes	No
1. Do you usually cough first thing in the morning in winter? (Count two or more coughs upon arising, or when you first go out of doors, or when you smoke the first cigarette of the day. Do not count clearing of throat.)	☐ 1	☐ 2	☐ 1	☐
	(COL. 11)		(COL. 12)	
	Yes	No	Yes	No
2. Do you usually cough during the day or night in winter? (Do not count an occasional cough.)	☐ 1	☐ 2	☐ 1	☐ 2
	(COL. 13)		(COL. 14)	
IF YOU ANSWERED "YES" TO QUESTION 1 OR 2, PLEASE ANSWER QUESTION 3.				
	Yes	No	Yes	No
3. Do you cough like this on most days or nights for as much as three months each year?	☐ 1	☐ 2	☐ 1	☐ 2

	MOTHER (or female guardian)	FATHER (or male guardian)
4. Do you usually bring up phlegm (thick fluid) from your chest first thing in the morning in winter? (Count phlegm whether swallowed or expelled, upon arising, or when you first go out of doors, or when you smoke the first cigarette of the day. Do not count phlegm from nose.)	(COL. 15) Yes ☐ 1 No ☐ 2	(COL. 16) Yes ☐ 1 No ☐ 2
5. Do you usually bring up phlegm from your chest during the day or night in winter?	(COL. 17) Yes ☐ 1 No ☐ 2	(COL. 18) Yes ☐ 1 No ☐ 2
IF YOU ANSWERED "YES" TO QUESTION 4 OR 5, PLEASE ANSWER QUESTION 6.		
6. Do you bring up phlegm like this on most days for as much as three months each year?	(COL. 19) Yes ☐ 1 No ☐ 2	(COL. 20) Yes ☐ 1 No ☐ 2
7. Do you get short of breath walking on level ground at an ordinary pace?	(COL. 21) Yes ☐ 1 No ☐ 2	(COL. 22) Yes ☐ 1 No ☐ 2
8. How many cigarettes do you usually smoke now?	(COL. 23)	(COL. 24)
Never smoked	☐ 1	☐ 1
Ex-smoker	☐ 2	☐ 2
Less than 1/2 pack per day (1-5 cigarettes)	☐ 3	☐ 3
About 1/2 pack per day (6-14 cigarettes)	☐ 4	☐ 4
About 1 pack per day (15-25 cigarettes)	☐ 5	☐ 5
About 1-1/2 packs per day (26-34 cigarettes)	☐ 6	☐ 6
About 2 or more packs per day (35 or more cigarettes)	☐ 7	☐ 7

FATHER-MOTHER HEALTH QUESTIONS CONTINUED

	MOTHER (or female guardian)	FATHER (or male guardian)
9. At your job, are you now or have you been frequently exposed to irritating smoke, dust, or fumes? (Do not include neighborhood or home exposures.)	(COL. 25) Yes ☐ 1 No ☐ 2	(COL. 26) Yes ☐ 1 No ☐ 2
IF YOU ANSWERED "NO" TO QUESTION 9, SKIP THE NEXT THREE QUESTIONS BELOW.		
9a. If the answer to question 9 is "yes," what kind of irritant were you exposed to? (For example: coal dust, cutting oils, asbestos, mine dust, smelter fumes, raw cotton dust.)		
9b. If the answer to question 9 is "yes," what kind of work did you perform in this job? (For example: miner, maintenance, assembly line, supervisor.)		
9c. If the answer to question 9 is "yes," how long were you exposed?	(COL. 27)	(COL. 28)
Less than 1 year	☐ 1	☐ 1
1 to 5 years	☐ 2	☐ 2
6 to 10 years	☐ 3	☐ 3
More than 10 years	☐ 4	☐ 4
DO NOT MARK THESE BOXES	(COL. 29) Yes ☐ 1 No ☐ 2	(COL. 30) Yes ☐ 1 No ☐ 2

(COL. 79-80) 0 2

10. Where did you live the longest when you were 0 to 20 years of age? (Check only one choice for each parent.)	MOTHER (or female guardian) (COL. 31)	FATHER (or male guardian) (COL. 32)
Present city, present neighborhood	☐ 1	☐ 1
Present city, different neighborhood	☐ 2	☐ 2
Different city or area of more than 200,000 people	☐ 3	☐ 3
Different city or area of about 50,000 to 200,000 people	☐ 4	☐ 4
Different city or area - A small city or town	☐ 5	☐ 5
Different city or area - A rural area or farm	☐ 6	☐ 6

11. Where did you live the longest when you were 21 to 30 years of age? (Check only one choice for each parent.)	(COL. 33)	(COL. 34)
Not yet 21 years old	☐ 1	☐ 1
Present city, present neighborhood	☐ 2	☐ 2
Present city, different neighborhood	☐ 3	☐ 3
Different city or area of more than 200,000 people	☐ 4	☐ 4
Different city or area of about 50,000 to 200,000 people	☐ 5	☐ 5
Different city or area - A small city or town	☐ 6	☐ 6
Different city or area - A rural area or farm	☐ 7	☐ 7

12. Where did you live the longest after you were 31 years of age? (Check only one choice for each parent.)	(COL. 35)	(COL. 36)
Not yet 31 years old	☐ 1	☐ 1
Present city, present neighborhood	☐ 2	☐ 2
Present city, different neighborhood	☐ 3	☐ 3
Different city or area of more than 200,000 people	☐ 4	☐ 4
Different city or area of about 50,000 to 200,000 people	☐ 5	☐ 5
Different city or area - A small city or town	☐ 6	☐ 6
Different city or area - A rural area or farm	☐ 7	☐ 7

B. QUESTIONS CONCERNING THE HEALTH OF CHILDREN 12 YEARS OF AGE OR YOUNGER WHO LIVE IN THE HOUSEHOLD

[illegible] 2

WRITE AT THE HEAD OF EACH COLUMN THE NAME OF EACH CHILD 12 YEARS OF AGE OR YOUNGER. → THEN, IN EACH CHILD'S COLUMN CHECK ONE BOX TO ANSWER EACH OF THE FOLLOWING QUESTIONS ↓	(COL. 9-10) ☐☐ Name of 1st child	(COL. 17-18) ☐☐ Name of 2nd child	(COL. 25-26) ☐☐ Name of 3rd child	(COL. 33-34) ☐☐ Name of 4th child	(COL. 41-42) ☐☐ Name of 5th child	(COL. 49-50) ☐☐ Name of 6th child	(COL. 57-58) ☐☐ Name of 7th child	(COL. 65-66) ☐☐ Name of 8th child	PLEASE CHECK BOX BELOW IF THERE ARE MORE THAN 8 CHILDREN 12 YEARS OF AGE OR YOUNGER.
1. Has your child been treated by a doctor since September 1977 for pneumonia?	(COL. 11) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 19) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 27) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 35) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 43) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 51) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 59) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 67) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 73) ☐ 2
2. Has your child been treated by a doctor since September 1977 for an attack of croup?	(COL. 12) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 20) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 28) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 36) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 44) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 52) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 60) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 68) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	
3. Has your child been treated by a doctor since September 1977 for an attack of bronchitis, bronchiolitis, or other deep chest infection?	(COL. 13) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 21) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 29) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 37) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 45) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 53) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 61) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 69) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	
4. Has your child been in the hospital since September 1977 for one of the illnesses mentioned in questions 1, 2, or 3 above?	(COL. 14) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 22) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 30) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 38) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 46) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 54) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 62) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	(COL. 70) ☐ No 1 ☐ Yes, once 2 ☐ Yes, twice 3 ☐ Yes, more than twice 4	
5. What is the full name and address of the doctor who takes care of your child or, if you take your child to a clinic for medical care what is the name and address of the clinic?	(COL. 15-16)	(COL. 23-24)	(COL. 31-32)	(COL. 39-40)	(COL. 47-48)	(COL. 55-56)	(COL. 63-64)	(COL. 71-72)	(COL. 79-80) [illegible]

III. GENERAL QUESTIONS CONCERNING THE HOUSEHOLD

(CARD 4)

(COL. 9)

1. What educational level has been completed by the head of the household? (Check one box only.)

☐ 1 Elementary school
☐ 2 Part of high school
☐ 3 High school graduate
☐ 4 Part of college
☐ 5 College graduate
☐ 6 Graduate school
☐ 7 Other ____________ (please specify)

(COL. 10)

2. What is the present employment status of the mother (or female guardian)? (Check one box only.)

☐ 1 Housewife or employed at home
☐ 2 Employed outside the home (full or part time)
☐ 3 Currently unemployed but usually employed outside the home.
☐ 4 Other ____________ (please specify)

(COL. 11)

3. What is the race of the family?

☐ 1 Indian
☐ 2 Mexican-American or Spanish American
☐ 3 Negro
☐ 4 Oriental
☐ 5 White
☐ 6 Other

(COL. 12)

4. How many times have you and your family changed living quarters during the last 5 years? (Check one box only.)

☐ 1 Zero
☐ 2 One
☐ 3 Two
☐ 4 Three
☐ 5 Four
☐ 6 Five or more

(COL. 79-80)
0 4

(COL. 13-14)

5. How long has the family lived in your present city or town? (Check one box only.)

☐ Less than 1 year
01
☐ 1 year
02
☐ 2 years
03
☐ 3 years
04
☐ 4 years
05
☐ 5 years
06
☐ 6 years
07
☐ 7 years
08
☐ 8 years
09
☐ 9 years
10
☐ 10 years
11
☐ 11 years
12
☐ 12 years or more
13

(COL. 15)

6. How many rooms are there in your living quarters? (Do not count bathrooms, porches, balconies, foyers, halls or half-rooms.) (Check one box only.)

☐ One
1
☐ Two
2
☐ Three
3
☐ Four
4
☐ Five
5
☐ Six
6
☐ Seven
7
☐ Eight
8
☐ Nine or more
9

(COL. 16)

7. Do you have air conditioning in your living quarters? (Check one box only.)

☐ No
1
☐ Yes, window only
2
☐ Yes, central
3

IV. CENSUS OF HOUSEHOLD

(CARD 5-19 - ONE CARD PER PERSON)

Names of persons living in the household		Asthma among persons living in the household (Check the appropriate box or boxes for each person.)				Chronic heart or lung disease among persons living in the household (Check "yes" for each person only if chronic heart or chronic lung disease was ever diagnosed by a doctor).				Sex of persons living in the household (Check below one appropriate box for each person.)		Age of persons living in the household Complete year of birth and age for each person as indicated below.		Position in family of persons living in the household (Check below one appropriate box for each person)				
(Write in the column below the first and [illegible] name of each person living in the household.)		Has this person ever had asthma diagnosed by a doctor?		Has this asthma been active in the past two years?		Chronic heart disease		Chronic Lung disease										
(COL. 9-10)	(COL. 11-29)	Yes	No (COL. 30)	Yes	No (COL. 31)	Yes	No (COL. 32)	Yes	No (COL. 33)	Male	Female (COL. 40)	Year of birth	Age in years at last birthday (COL. 41-42)	Head of household	Spouse (COL. 43)	Child	Other	(COL. 79-80)
01.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	05
02.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	06
03.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	07
04.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	08
05.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	09
06.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	10
07.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	11
08.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	12
09.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	13
10.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	14
11.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	15
12.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	16
13.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	17
14.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	18
15.		☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐ 1	☐ 2	☐☐☐☐	☐	☐ 1	☐ 2	☐ 3	☐ 4	19

THANK YOU FOR YOUR COOPERATION